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RUDOLPH MATAS

The Editorial Board notes with profound sorrow the death on September 23, 1957, at the age of 97, of Dr. Rudolph Matas, a charter member and third president of The American Association for Thoracic Surgery and a member, from its inception in 1932, of the Advisory Editorial Board of the JOURNAL OF THORACIC SURGERY. Thus passes from the American and international scene a great figure in Surgery, respected, revered, and beloved by all fortunate enough to have known him during his long and distinguished career. It can be said of Dr. Matas that in the depth of his medical and surgical knowledge, in the facility with which he presented that knowledge to others, in several languages, if need be, and in the beautiful style of his spoken and written word, he was truly unique, unequaled, and unexcelled. His loss as a valued associate on the Editorial Board and as a sympathetic and loyal friend is mourned by all his fellow members on the Board.

Original Communications

RECONSTRUCTION OF THE ESOPHAGUS WITH SEGMENTS OF THE COLON

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GEORGE H. A. CLOWES, JR., M.D.
CLEVELAND, OHIO

ESOPHAGEAL replacement utilizing the colon to create an antethoracic subcutaneous esophagus was first used by Kelling and Hacker in the early 1900's. Although, in a review of the literature from 1911 to 1934, Ochsner found only 20 cases of subcutaneous colon transplants,[15] in the past few years there has been a revival of this method in Europe. Our unsatisfactory results in using the stomach to restore alimentary continuity, following resection for carcinoma, led us to investigate the technique of replacing the esophagus with the colon.

It is the purpose of this paper to present our clinical and experimental observations on the efficacy of using segments of the colon within the thorax to restore esophageal continuity following resection in both benign and malignant disease. While this over-all approach to reconstructing the esophagus is not new, certain recent advances of medicine appear to make this a reasonable approach to this intriguing problem.

EXPERIMENTAL PROCEDURE

To evaluate the reconstruction of the esophagus by implanted colon, a series of 6 normal dogs were operated upon. Through a thoracoabdominal incision, the esophagus was resected from the inferior pulmonary vein to the cardioesophageal junction. The opening in the stomach was inverted. An appropriate segment of transverse colon was interpolated in isoperistaltic fashion between the esophagus above and a separate opening in the fundus of the stomach. A colocolostomy reconstituted the colon. Pyloromyotomy or pyloroplasty provided adequate drainage of the vagotomized stomach. These dogs were sacrificed at varying periods up to 9 months following surgery. In a comparative series, 5 dogs had a resection of the distal end of the esophagus and an esophagogastrostomy performed. Bilateral vagotomy and pyloromyotomy or pyloroplasty were done. The diaphragm was sutured to the stomach so that a considerable portion of the latter lay well within the thorax. These dogs were then sacrificed after varying periods of time up to 3 months following surgery.

EXPERIMENTAL RESULTS

The lower end of the esophagus, the transplanted colon, and the entire stomach were removed for post-mortem examination. In none of the animals

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Read at the Thirty-seventh Annual Meeting of the American Association for Thoracic Surgery at Chicago, Ill., May 4-7, 1957.

in this group was there evidence of esophagitis, colitis, or gastritis (Fig. 1). Post-mortem examination in the second group of animals showed complete absence of the mucosa in the lower end of the esophagus above the esophagogastric junction. There were 3 instances of diffuse ulceration of the fundus which lay within the thoracic cavity. The mucosa for the most part, extending up to 3 to 4 inches above the cardioesophageal junction, was markedly flattened with evidence of esophagitis and ulceration (Fig. 2).

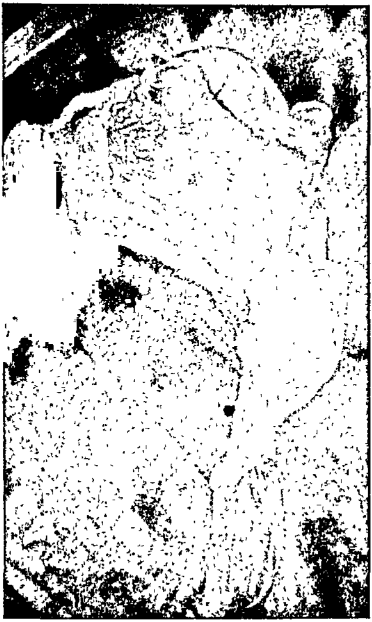


Fig. 1.

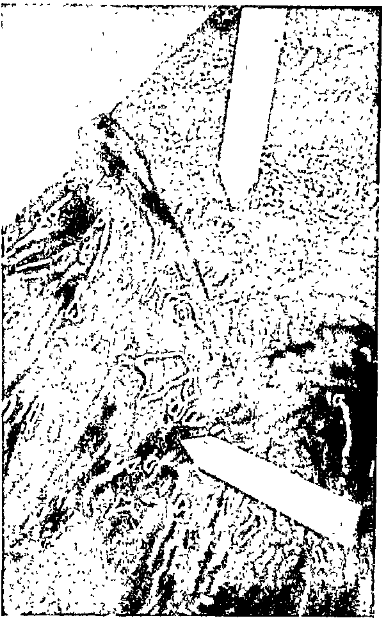


Fig. 2.

Fig. 1.—Post-mortem examination of esophagus, interposed colon, and stomach in a dog sacrificed at 9 months. There is no evidence of esophagitis, colitis, or gastritis.

Fig. 2.—Post-mortem examination following an esophagogastrostomy. There was diffuse inflammation in the stomach and ulceration of the esophageal mucosa.

CLINICAL OPERATIVE TECHNIQUE

The esophagus was reconstructed in one stage with the exception of one child in whom a delayed reconstruction was performed at the age of one year. Two separate operating teams have been employed. Satisfactory preoperative nutrition and chemical balance were obtained by either preliminary feedings through a small polyethylene tube passed into the stomach at the time of the esophagoscopy or a tube gastrostomy. The colon was prepared for 2 to 4 days with neomycin (1 Gm., 4 times daily), Fleets phosphosoda (½ oz., twice daily), and cleansing enemas. Although the technique varied depending upon the portion of the esophagus to be resected, the principles of using the colon for replacement remained the same in all instances.

Benign and malignant lesions in the cervical region are operated upon with the patient in the supine position. If a thoracotomy is deemed necessary, the right side of the chest is elevated to a 45-degree angle. The esophagus is exposed in the neck through an incision along the anterior border of the right sternocleidomastoid muscle. The strap muscles are divided transversely over the thyroid gland and the latter retracted medially. The esophagus is isolated just above the manubruim and dissected upward, care being taken to identify and preserve the recurrent laryngeal nerve.

Concomitantly the abdomen is opened through an upper midline incision from the xiphoid process to well below the umbilicus. The lesser peritoneal cavity is entered midway along the greater curvature to the stomach and the gastrocolic omentum divided to the hepatic flexure. The lateral avascular peritoneal attachments of the ascending colon and cecum are severed and the entire right colon reflected medially. The appendix is removed whenever the cecum is to be used for the esophageal anastomosis. The middle, right, and iliocolic arteries are identified. The right colic artery is occluded close to its origin with a bulldog clamp. This allows observation of the adequacy of the middle colic artery to supply the cecum through the marginal arteries. The gastrocolic omentum is divided distally to the splenic flexure while the right colic vessel is occluded so that no operating time is wasted. If the circulation to the cecum appears to be adequate, the right colic and a branch of the iliocolic arteries are ligated near their origins and the ileum transected at the iliocolic junction. Following closure of the opening in the cecum, the right half of the colon is placed on the anterior chest wall to determine its adequacy for esophageal reconstruction. The mesentery on this side is divided down to the origin of the middle colic vessels. A tunnel is created in the anterior mediastinum just posterior to the periosteum of the sternum from the xiphoid process to the manubrium by blunt dissection from below and above (Fig. 3). The proximal end of the colon is passed into the neck through this tunnel using a long clamp inserted from above and down to the abdomen. To widen the thoracic inlet, the upper sternum has been split in four operations. Having determined that the cecum will adequately reach the neck without compromising its blood supply, the colon segment is isolated by dividing the transverse colon. Its mesentery is separated down to the middle colic vessels. Thus, the entire segment of the bowel is suspended upon a vascular pedicle comprised of only the middle colic artery and vein. The esophagus is transected above the lesion. The cervical esophageal colon anastomosis is performed end-to-end by the method of Sweet[21] with two or three layers of interrupted fine silk (Fig. 4). If the distal esophagus is not resected at this time, a catheter is inserted into this section and brought out through a stab wound to create a mucus fistula. Simultaneously, an opening is made on the anterior wall of the stomach and the distal end of the colon anastomosed to this organ. Either a pyloromyotomy or pyloroplasty may be performed. If a tube gastrostomy for preoperative feeding purpose has not

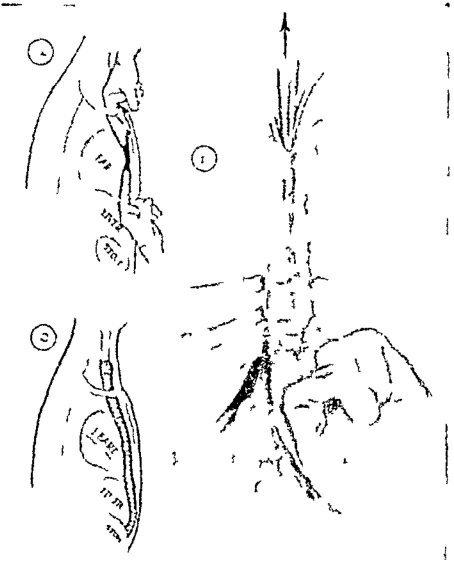


Fig 3.—Drawing of anterior mediastinal tunnel created by blunt dissection from xiphoid the manubrium

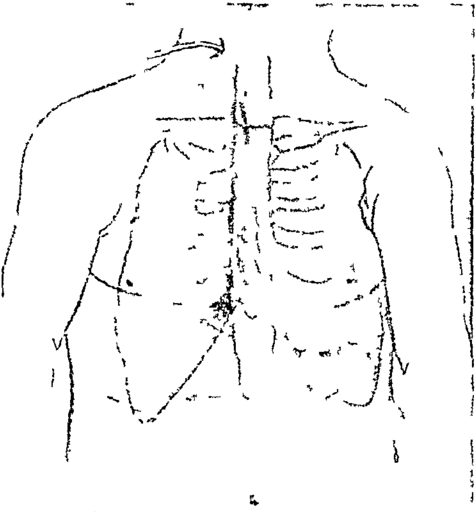


Fig 4.—Drawing depicting completed anterior mediastinal colon transplant

been done, this is placed to decompress the stomach in the early postoperative period. It may then be used for subsequent feedings until such time as the patient is able to eat normally. This maneuver obviates the necessity for an indwelling nasogastric tube. The continuity of the lower gastrointestinal tract is established with an end-to-end iliocolostomy.

Intrathoracic esophageal lesions above the aortic arch are approached through a right thoracotomy and upper right paramedian incision. The patient is positioned on the operating room table at a 45-degree angle. This allows the thoracic team to perform an adequate resection of the esophagus and invert the lower end. Simultaneously, with the patient in this position, the colon can be mobilized by the abdominal surgeon, if he will perform the operation while standing on the left side of the patient. Here the colon is delivered into the chest through an opening in the diaphragm anterior to the right lobe of the liver, and is placed in front of the hilum of the lung. The remainder of the operating technique is similar to that described previously (Fig. 5).

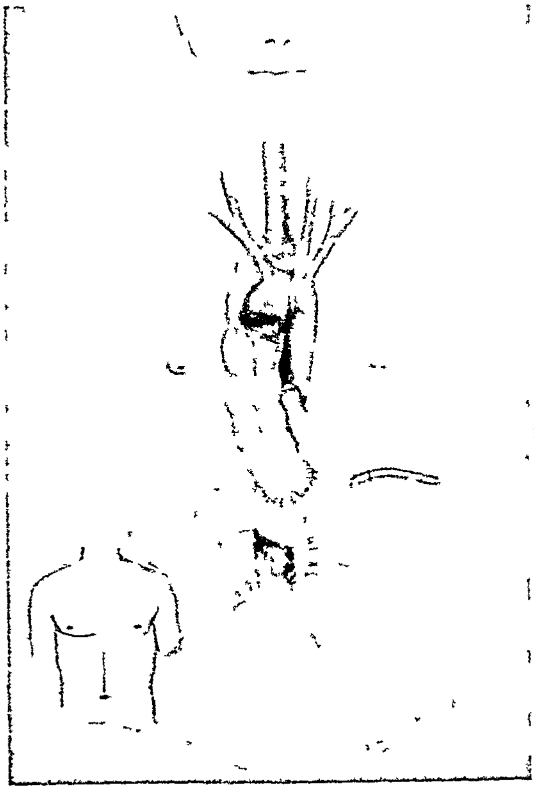


Fig. 5.

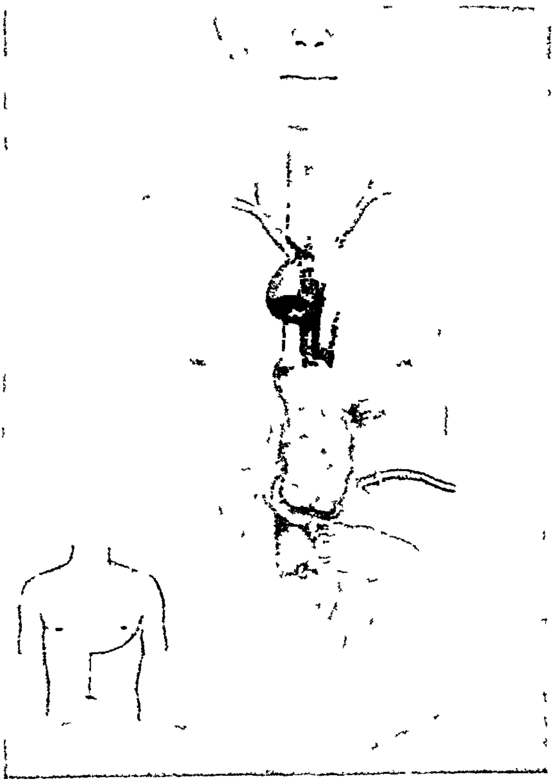


Fig. 6

Fig. 5.—Drawing of completed intrathoracic colon transplant for lesions above the aortic arch.

Fig. 6.—Drawing of procedure for establishing upper gastrointestinal continuity following resection of intrathoracic lesions below the aortic arch.

Intrathoracic esophageal lesions below the aortic arch are approached through separate left abdominal and thoracic incisions. It has not been necessary to cut across the costal margin. The diaphragm can be transected from the hiatus outward to allow ample room for resection of the distal esophagus, and cardia of the stomach if necessary. In this situation it may not be necessary to mobilize the entire right colon. The transverse colon is usually of sufficient length to substitute for the resected esophagus. Following an adequate resection of the lesion, the proximal portion of the bowel is passed into the left chest. The isoperistaltic esophagocolon anastomosis is performed in the manner previously described. The bowel is replaced in the bed of the resected esophagus and the entire area repleuralized. If the stomach is normal, the opening in the cardia is closed from above and the distal end of the colon is anastomosed to the stomach anteriorly. However, if a resection of a portion of the stomach is necessary, the distal end of the colonic segment may be anastomosed end to end to the remaining portion of the stomach, a pyloroplasty and a tube gastrostomy are performed. A colocolostomy completes the operative procedure (Fig. 6). The average operating time on all of the patients was 3½ hours. Culture of the colon is done at the time of surgery to perform drug sensitivity studies, and the appropriate antibiotic is given postoperatively. The gastrostomy tube is placed on continuous suction for a period of 3 or 4 days. This method of gastric decompression makes the use of an indwelling nasogastric tube unnecessary. The patient is given nothing by mouth for at least 10 days. He is fed through the gastrostomy tube beginning on the sixth postoperative day. Oral fluids are allowed on the tenth postoperative day. The diet is gradually increased until the individual is eating in a normal manner.

RESULTS AND COMPLICATIONS

To date, the colon has been used to reconstruct the esophagus in 28 patients. As depicted in Table I, 23 had carcinoma, and the remaining 5 benign disease. The 7 deaths in the immediate postoperative period were in patients with carcinoma above the aortic arch. Five additional patients have died in the interim from a progression of their disease or coronary occlusion. At the moment, 16 patients are living.

Table I. Results of Esophageal Replacement With Colon—Mortality

LESION AND LOCATION	NUMBER	DEATHS			LIVING	
		IMMEDIATE POSTOPERATIVE	LATE	AVERAGE SURVIVAL TIME (MONTHS)	ALIVE	SURVIVAL TIME (MONTHS)
Carcinoma above arch	12	7	5	8.2 (2-18)		
Carcinoma below arch	11	0	0		11	3-36
Stricture (corrosive) total esophagus	1	0	0		1	5
Stricture (corrosive, atresia and esophagitis) below arch	4	0	0		4	4-21

One patient died of generalized peritonitis on the sixth postoperative day due to dehiscence of his cologastrostomy. This may have been related to gastric distention which resulted from failure to perform a pyloroplasty and a tube gastrostomy.

Two other deaths were preventable. One elderly man died during the night of a spontaneous pneumothorax. Another patient died following disruption of the cervical and thoracic incisions. By mistake, the sutures were removed on the sixth day, post-mortem examination disclosed leakage of gastric contents into the posterior mediastinum from a distended lower esophageal stump. Here again, a tube gastrostomy to decompress the stomach had been omitted.

At the outset of this present study, it was our feeling that in nonresectable carcinomas the patient might be given palliation if the lesion was bypassed with the colon. Upon establishing a satisfactory swallowing mechanism, the primary lesion could be irradiated. This was done in 4 patients with extensive high carcinomas. None of these patients left the hospital, and in none was roentgen therapy instituted. Even though they obtained an adequate caloric intake through the gastrostomy tube, wound healing was poor. Ultimate breakdown of one or more of the anastomoses occurred with a fatal outcome in each of these patients.

Five individuals died 2 to 18 months postoperatively due to metastasis or heart disease. All of these patients could swallow in a normal manner and initially gained weight. At the moment, 16 patients are living who have been followed from 3 to 36 months. One hundred per cent of the patients had a normal appetite with normal ingestion of food. Ninety-four per cent had an improved nutritional status and have gained weight. Hemoglobin, hematocrit, and serum proteins were normal in all individuals at one time in the postoperative period.

The commonest nonfatal complication was diarrhea. This occurred in half of the surviving patients and in all of the experimental animals. It is associated with ingestion of food. Fortunately, this usually persisted for only a few weeks.

Three of the earlier cases had varying degrees of gastric retention. The use of a pyloromyotomy or a pyloroplasty in association with suction through a gastrostomy has prevented this occurrence.

Empyema without a demonstrable anastomotic leak occurred in 2 patients. They responded satisfactorily to frequent early aspiration and subsequent open drainage.

In 3 patients, because the middle colic vessels arose from superior mesenteric artery and vein to the extreme right of the midline, it was necessary to use the distal end of the bowel for the esophageal anastomosis. All of these patients complain of nocturnal regurgitation. Reverse peristalsis can be demonstrated in these segments fluoroscopically.

Atelectasis has been a disturbing complication in resecting high intrathoracic carcinomas. In 4 elderly patients, collapse of the right lung occurred soon after surgery. All responded satisfactorily to bronchoscopy and tracheostomy.

Narrowing of the esophago-colon anastomosis in the neck occurred in 2 patients. One followed closure of a fistula and the other was due to angulation. Revision was successfully carried out in both instances.

CASE REPORTS

CASE 1.—*Stricture of the entire esophagus* (Fig. 7): B. S., a 30 month old Negro male child, was admitted to Cleveland City Hospital on Nov. 20, 1956, because of persistent regurgitation of food of 2 weeks' duration. No history of ingestion of corrosive material could be obtained from the mother. Barium swallow on Nov. 23, 1956, revealed two areas of narrowing of the esophagus, one at the midportion and the other 2 cm. proximal to the cardia. Esophagoscopy on Nov. 27, 1956, demonstrated narrowing of the lumen at 13 cm. The mucosa appeared reddened and friable. No attempt was made to pass the esophagoscope beyond this level, but biopsy was obtained which subsequently proved to be noncontributory. Several hours following esophagoscopy, the baby developed crepitation of the neck. A roentgenogram of the chest revealed subcutaneous emphysema and a pneumomediastinum. He was immediately taken to surgery where a superior mediastinotomy and a tube feeding gastrostomy was performed. Aside from the subsequent single aspiration of 120 c.c. of straw-colored fluid from the left hemithorax, his recovery was uneventful. Progress x-ray studies showed narrowing of the esophagus, and because of the unknown etiology, a secondary esophagoscopy was undertaken on Jan. 14, 1957, which demonstrated a white scarred ring 11 cm. from the incisor teeth. A No. 5 esophagoscope could not be passed beyond the stricture. No attempt was made to biopsy the area because of the previous unfortunate episode. The child was prepared for surgery in the usual manner and operated upon Jan. 30, 1957. Because the stricture began in the cervical region, we deemed it advisable to perform a two-stage procedure upon this child, primarily to interpolate the colon between the normal esophagus in the neck and the stomach through a mediastinal tunnel without opening the chest, and subsequently to resect the entire remaining esophagus via a right thoracotomy. The operation was performed with the child in the supine position. The esophagus was exposed in the neck through an incision along the anterior border of the

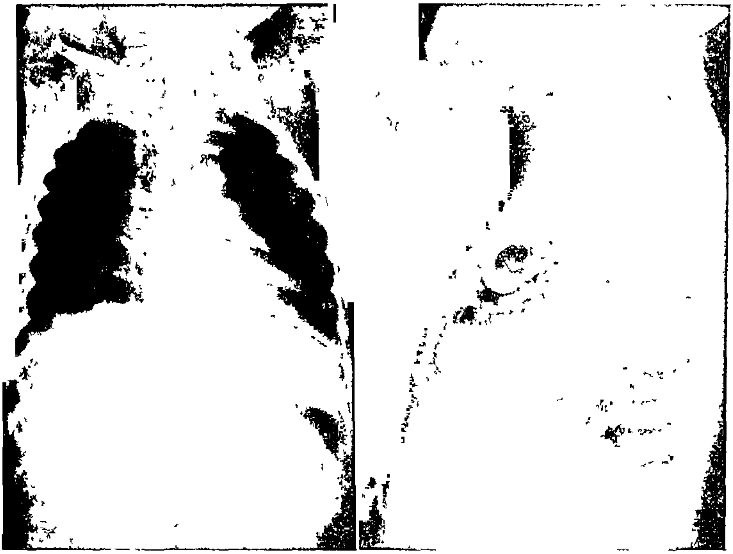


Fig. 7.—Roentgenogram on right shows a long esophageal stricture. Postoperative film 5 months following colon transplant through anterior mediastinal tunnel.

right sternocleidomastoid muscles. A V-shape segment of the sternum was removed to increase the anterior posterior diameter at the thoracic inlet. This maneuver allows the colon to be displaced into the neck without constriction at this level. The esophagus was transected at the level of the manubrium and a small catheter inserted into the distal end in an attempt to create an external mucus fistula. This was brought out through a stab wound lateral to the original incision. Through a separate abdominal incision, the colon was mobilized from the cecum to the splenic flexure in the manner previously described. A tunnel was created in the anterior mediastinum from below and above. The cecum was displaced into the neck through this tunnel and end-to end esophagocolostomy was performed 2 cm. below the inferior pharyngeal constrictor muscles. The distal cut end of the colonic segment was anastomosed to the stomach and end-to end iliocolostomy established lower gastrointestinal continuity. The pyloroplasty completed the operation.

His postoperative course was most gratifying. Gastrostomy feedings were begun on the seventh day and oral feedings on the eleventh. He was discharged on February 20, at which time he was eating chopped foods normally.

He was readmitted to the hospital on March 26. The mother stated that he had experienced some difficulty in swallowing in the previous 2 weeks. X-ray studies of the esophagus and transplanted colonic segment demonstrated narrowing of the esophagocolostomy. The barium however easily traversed this area into the stomach. Hemoglobin, hematocrit, and serum proteins were normal.

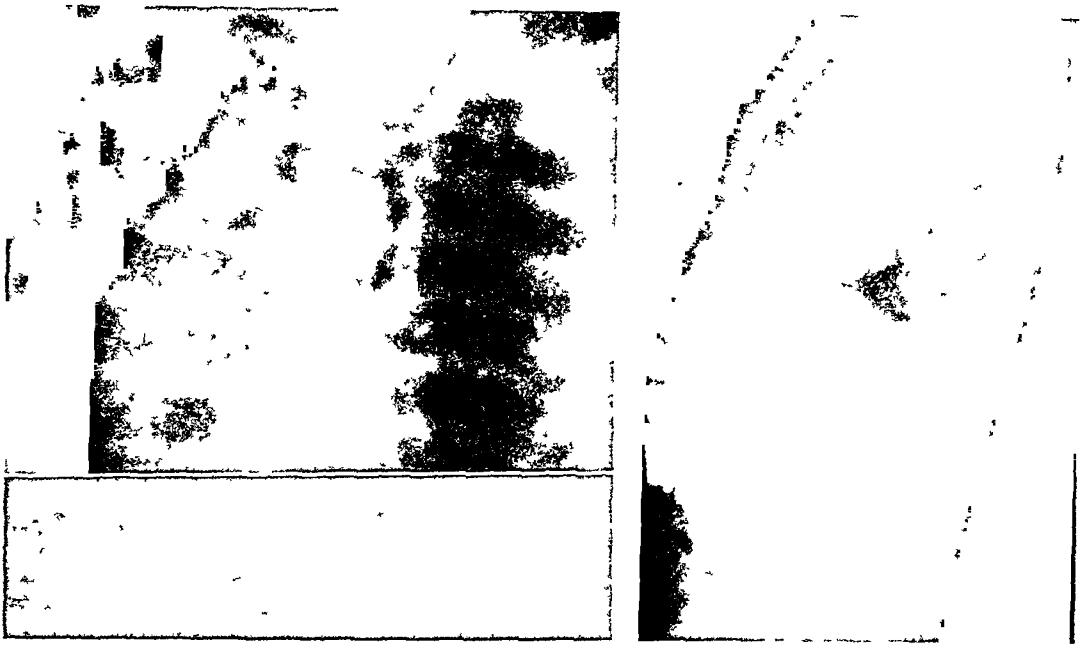


Fig. 8.—Delayed reconstruction of esophagus for tracheosophageal fistula with atresia. Anterior mediastinal colon transplant 21 months postoperatively.

Case 2.—*Delayed reconstruction of esophagus in tracheoesophageal fistula with atresia* (Fig. 8): Baby F. was operated upon July 22, 1954, at the age of 4 days for a tracheoesophageal fistula with atresia. After ligation of the fistula, the lower end of the esophagus was resected. A feeding gastrostomy and cervical esophagostomy were performed.

Due to the extreme diligence in the care of this child at the age of one year he was felt to be a suitable candidate for a reconstruction of the esophagus. From previous experience in the use of the colon for esophageal replacement following resections for carcinoma, we felt that this segment of bowel could be used for almost a total esophagoplasty.

He was operated upon Aug. 27, 1955. With the baby in the supine position ,the abdomen was opened through a long upper midline abdominal incision. The colon was mobilized from the cecum to the splenic flexure. It was transected proximally and distally after an adequate length for esophageal reconstruction had been determined.

The cervical esophagostomy was dissected free and a tunnel created in the anterior mediastinum just posterior to the periosteum of the sternum. The proximal end of the colon segment, which was suspended on a pedicle of the middle colic artery and vein, was passed through the mediastinal tunnel into the neck. An esophagocolostomy and cologastrostomy were performed concomitantly. A colocolostomy reunited the lower bowel.

His postoperative course was uneventful except for the development of a cervical fistula. This healed readily and he was eating normally at the time of his discharge.

On Dec. 15, 1955, he was admitted to the hospital because of difficulty in swallowing. It was obvious that he had a stricture at the esophagocolostomy but, because of severe diarrhea and an elevated temperature, a revision of his anastomosis was postponed. On December 18, he suddenly became cyanotic and dyspneic necessitating an emergency exploration of the neck. Although it was our intention to perform an immediate tracheostomy, the dilated upper esophageal segment, which was adherent to the skin, was opened. Marked narrowing of the colon was found at the thoracic inlet. The stricture was manually dilated with a clamp until an adequate opening was obtained. The esophagus was closed and a tracheotomy performed. He made a satisfactory recovery and today is a healthy active child.

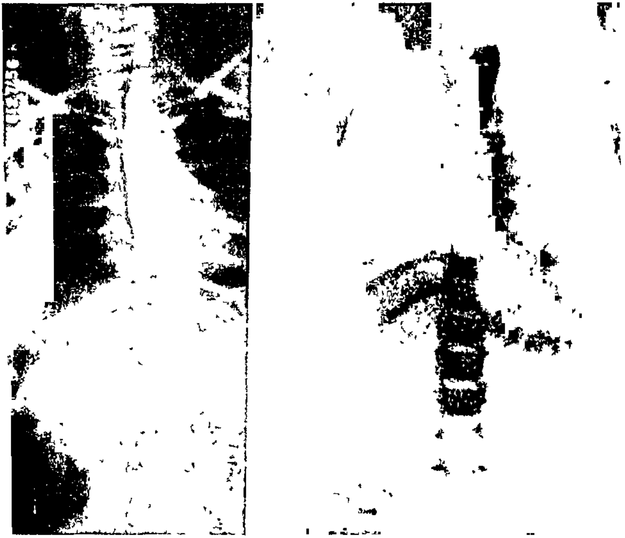


Fig. 9.—Colon transplant for lower esophageal stricture following ingestion of muriatic acid

Case 3.—*Stricture of the lower esophagus following ingestion of corrosives* (Fig. 9): J. S., a 6-year-old boy, was admitted to University Hospitals on Oct. 19, 1953, shortly after swallowing muriatic acid. An emergency tracheostomy was performed because of difficulty in breathing. He was placed on cortisone and antibiotics. Barium swallow one week after admission demonstrated a stricture at the lower end of the esophagus and pyloric obstruction. Bouginage of the esophagus was successfully carried out but the pyloric obstruction persisted. Resection of the distal end of the stomach was done on Dec. 10, 1953, a Bilroth I type of procedure being performed. Despite repeated esophageal dilatations, dysphagia and vomiting persisted. Neuropsychiatric therapy was instituted because of absence of roentgenographic evidence of pyloric obstruction. Narrowing of the distal end of the esophagus was repeatedly evident by x-ray study and esophagoscopy. On several occasions it was necessary to remove a bolus of food from the esophagus. In June, 1955, x-ray studies of the esophagus and stomach showed 90 per cent gastric retention at the end of 6 hours. He was admitted to St. Alexis Hospital where revision of the gastroduodenostomy was done on July 1, 1955. The lower end of the esophagus was definitely narrowed but there appeared to be normal passage of barium through this area. Following this he was relieved of his symptoms only temporarily. He was admitted to City Hospital on July 7, 1956, for consideration of resection of the esophageal stricture. Roentgenograms showed a persistent narrowing at the lower third of the esophagus with an adequate gastroduodenal stoma. On July 12, 1956, the lower end of the esophagus was resected and the transverse colon interpolated in an isoperistaltic manner between the esophagus and the stomach. His postoperative course was uneventful except for transient rise in fever in his second postoperative day. Blood culture was done at this time and it was positive for *Aerobacteraerogenes.* Tube feedings were begun on the seventh postoperative day and oral feedings on the tenth day. He was discharged on the sixteenth postoperative day at which time he was eating a normal diet. The gastrostomy tube was removed just prior to his leaving the hospital. He was readmitted to the hospital on March 12, 1957, for follow-up studies. In the interim his mother stated that he was now able to eat everything and had no regurgitation. His only complaint was occasional abdominal cramps and diarrhea. Weight gain had been approximately 4 pounds in 2 months. X-ray studies showed slight delay in the passage of barium from the esophagus into the colonic segments but was otherwise normal. Blood studies performed at this time showed a hemoglobin of 12 Gm., white blood count of 6,780. Total proteins were 6.4, albumin 4.9, globulin 1.5 with an A/G ratio of 3.27.

CASE 4.—*Carcinoma of the distal end of the esophagus with invasion of the stomach* (Fig. 10): R. W., a 49-year-old Negro man, was admitted to Cleveland City Hospital on May 20, 1956, complaining of dysphagia of one year's duration. During the interim he had lost 40 pounds of weight. Esophagrams demonstrated an irregularity of the distal third f the esophagus with almost complete obstruction at this level. On esophagoscopy, a squamous-cell carcinoma was biopsied 40 cm. from the incisor teeth. Following satisfactory chemical and nutritional balance and the usual bowel preparation, the patient was operated upon Jan. 11, 1956. With the patient in the lateral position, the left chest was entered through the seventh interspace. The lesion was found to extend from a position approximately 9 cm. above the diaphragm down to and through the hiatus. The esophagus was mobilized from the aortic arch down to the diaphragm. The latter was incised anteriorly to the costal margin. Simultaneously through a left paramedium incision, the transverse colon was mobilized by the abdominal team. Since the lesion was found to be invading the stomach, the proximal two thirds of this organ was removed along with the lower esophagus. A section of the transverse colon on a pedicle comprised of only the middle colic vessels was brought up into the chest. The proximal end of the bowel was anastomosed to the esophagus about 4 cm. below the arch of the aorta. The distal end of the colonic segment was anastomosed end-to-end to the remaining portion of the stomach. A pyloromyotomy was done as was also a tube gastrostomy. Lower intestinal continuity was re-established with an end-to-end colocolostomy. Before closure of the chest, the segment of the colon in the thorax along with a proximal portion of the esophagus was entirely repleuralized. The duration of the operation was 4 hours, during which time he received 8 units of blood. Following extubation, he developed difficulty in breathing and it became obvious that the left lung was collapsed. A tube was inserted into the thorax anteriorly in the third interspace and, because of the tracheobronchial secretions, a tracheostomy was done. He responded satisfactorily to these measures and subsequently had an uneventful convalescence.

Pathologic sections demonstrated an extensive squamous-cell carcinoma of the lower end of the esophagus with invasion of the proximal portion of the stomach. The esophagus had been divided through normal tissue 2 cm. proximal to the lesion and the stomach divided 7 cm. distal to the lesion. The lymph nodes removed with the specimen were negative for neoplasm. He was discharged on the thirty-fifth postoperative day. Since leaving the hospital, he has gained 15 pounds and is back to work as a laborer in the steel mill. His appetite is normal and he has no abdominal distress and no regurgitation. X-ray studies reveal normal passage of barium through the upper gastrointestinal tract. Hemoglobin, hematocrit, and serum proteins are within normal limits.

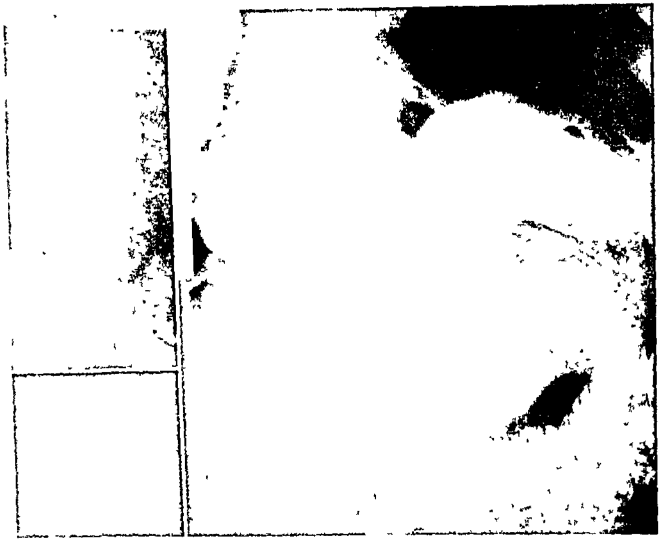


Fig. 10.—Carcinoma lower end of esophagus resected and colon transplant performed one year previously. Patient gained 15 pounds and works in a steel mill.

Case 5.—*Benign stricture of lower third of the esophagus due to esophagitis and hiatal hernia* (Fig. 11): H. K., a 55-year-old white man, was first seen in Polyclinic Hospital in February, 1956, because of hematemesis and tarry stools of 3 days' duration. X-ray studies of the upper gastrointestinal tract were interpreted as showing evidence of esophagitis, and hiatal hernia with a 2 cm. constriction above the left leaf of the diaphragm. Esophagoscopy showed a concentric narrowing of the lumen 29 cm. from the incisor teeth. A biopsy of this area was reported as chronic esophagitis. In view of the symptoms, he was operated upon Dec. 4, 1956. The lower end of the esophagus was hypertrophied and one could feel a firm indurated area just above the cardioesophageal junction. Due to the shortened esophagus, about 4 inches of the cardia was above the diaphragm. The stomach was opened longitudinally just below the cardioesophageal junction and the incision continued up into the esophagus through the stricture. A frozen section of this segment disclosed no evidence of neoplasm. In view of this, the esophagus was closed transversely. The diaphragm was incised from the hiatus outward. A Foley catheter was inserted in the stomach and brought out through a stab wound in the left upper quadrant for decompression of the stomach in the early postoperative period. Because of the short esophagus, it was impossible to place the cardioesophageal junction below the level of the diaphragm. The postoperative course was uneventful. He was discharged December 19, eating a normal diet. X-ray studies of the esophagus and stomach just prior to discharge showed very little change in the pathologic entity. It was felt that an inadequate operation had been performed and that sooner or later this man would again get into difficulty and a different type of operative approach would be considered. He was readmitted to the hospital just 6 days later on December 24, because of gastrointestinal bleeding. The hemoglobin was 2.6 Gm. with a hematocrit of 7. The treatment consisted of Sippy diet and multiple transfusions. He was discharged markedly improved on Jan. 18, 1957, with a hemoglobin of 11 Gm. He was admitted to the hospital a third time on March 12, 1957, complaining of persistent lower thoracic and epigastric distress. The hemoglobin was 8 Gm. and he had a hematocrit of 24. Tarry stools had been present for 3 days.

In view of the symptoms of recurring upper gastrointestinal bleeding, it was felt that the lower esophagus should be resected, the stomach placed beneath the diaphragm, and the esophagus reconstructed with a segment of the transverse colon.

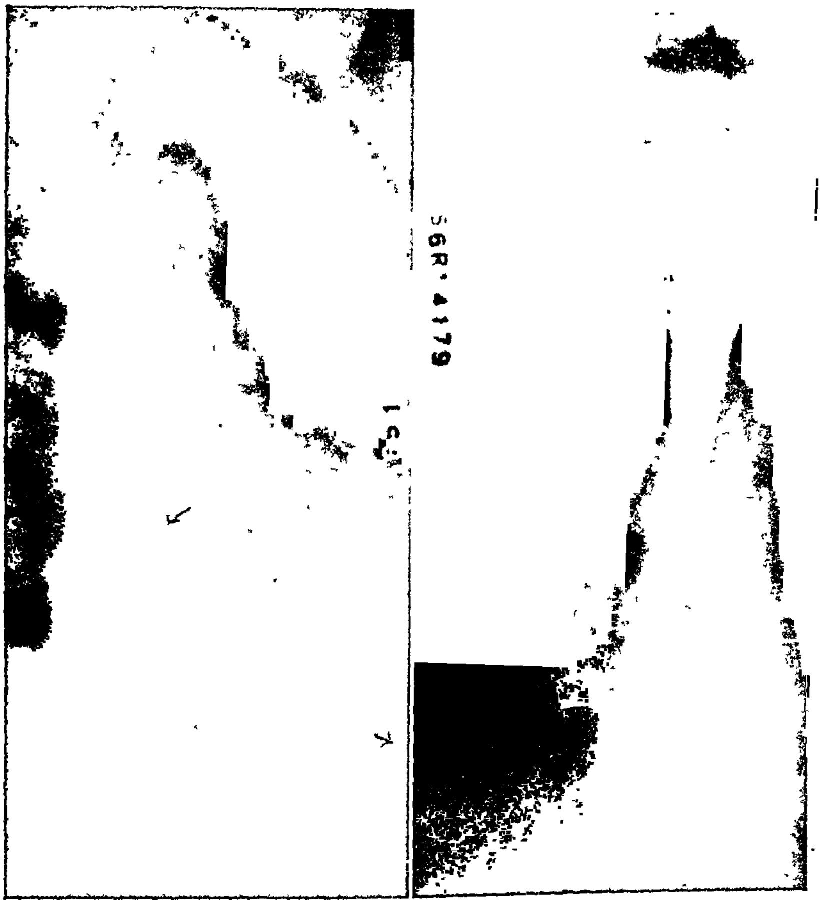


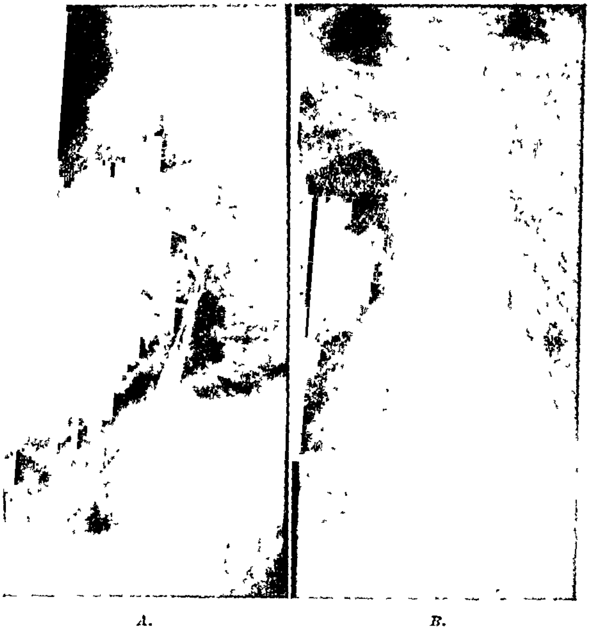
Fig. 11.—Esophagus reconstructed with the colon following resection of a stricture of lower end due to esophagitis and hiatal hernia. Previous Heinicke-Mikulicz procedure with a hiatal herniorrhaphy was unsuccessful.

This operative procedure was done on March 19, 1957. The abdomen and the left thorax were simultaneously opened. The lower end of the esophagus was resected and the cardia closed and isoperistaltic segment of transverse colon was interpolated between the esophagus and a separate opening at the fundus of the stomach. A colocolostomy, pyloroplasty, and a tube gastrostomy were performed.

The postoperative course was most gratifying. Gastrostomy feedings were begun on the sixth day and oral feedings on the tenth. On discharge from the hospital, he was eating satisfactorily and completely relieved of his symptoms.

Case 6.—*Carcinoma above the aortic arch* (Fig. 12): D. S., a 53-year-old Negro man, was admitted to Cleveland City Hospital on April 8, 1956, with a left lower lobe pneumonia. He stated that for 3 weeks he had had difficulty in swallowing solids. An esophagram demonstrated a lesion at the level of the aortic arch. On esophagoscopy, a squamous-cell carcinoma was biopsied 25 cm. from the incisor teeth.

He was operated upon April 20, 1956. With the patient positioned on the operating table at a 15 degree angle, the right thorax and abdomen were simultaneously opened. The esophagus was resected by the thoracic team from 4 inches above the azygos vein down to the diaphragm and the lower end inverted. A frozen section of the upper end of the esophagus was negative for carcinoma. At the same time the colon was mobilized from the cecum to the splenic flexure. It was severed proximally and distally and the cecum delivered into the chest through the diaphragm anterior to the liver. An esophagocolostomy and cologastrostomy established upper gastrointestinal continuity. An iliocolostomy, pyloroplasty, and tube gastrostomy completed the operative procedure.



A. *B.*

Fig. 12.—Esophagus reconstituted with the colon following resection of carcinoma above the arch.

A, Preoperative: Negro man, aged 54; carcinoma above aortic arch.

B, Postoperative: Resection with colon transplant anterior to right hilum. Patient living and well at 10 months.

His postoperative course was rather prolonged and stormy. He developed an incarcerated hernia which was repaired on the tenth day after operation. An empyema in the right thorax was drained on the sixteenth day. Following this, an esophagopleural cutaneous fistula was demonstrated. This healed in a short time and at the time of his discharge he was eating in a normal manner.

He subsequently gained weight and felt well until November, 1956. He returned to the out-patient clinic at this time complaining of difficulty in swallowing. Esophagoscopy revealed recurrent carcinoma near the esophagocolon anastomosis. (It is interesting that sections through this area at the time of surgery showed no evidence of neoplasm.) X-ray therapy was instituted with only temporary improvement. He died recently at another hospital. No post-mortem examination was performed.

DISCUSSION

Reconstruction of the esophagus has been a problem since the first successful resection in 1913 by Torek.[23] Initially antethoracic skin tubes enjoyed considerable popularity. However, the multiple stages with numerous failures due to strictures and fistulas caused this to be abandoned.[25] The stomach has been used subcutaneously and intrathoracically since the first successful reconstruction by Adams and Phemister in 1938,[1] following resection of a carcinoma of the lower third. Due to the work of Sweet[21] and Garlock[5] higher and higher transplantations have been performed. The stomach has distinct advantages—it is readily available, has a good blood supply, and can be mobilized easily. However, the development of peptic esophagitis, regurgitation of gastric contents into the pharynx in high resections, and the general unhappy state of many of these individuals in regard to their eating habits are distinct disadvantages. The development of esophagitis and gastritis in our experimental animals within a relatively short period of time lends support to the thesis that the stomach is not a good substitute for the esophagus.

Subcutaneous jejunal transplantation was first used successfully by Roux in 1907.[19] In recent years, intrathoracic jejunal replacement of the esophagus has received considerable impetus from the work of Rienhoff,[16] Harrison,[8] and Robertson.[17] However, the short vascular arcades make it difficult to obtain a long healthy segment.

Although inert plastic tubes could serve as an esophageal bridge, from our own personal experiences in the laboratory,[13] we do not believe this should be done in man.

The use of the colon to replace the esophagus is not a new idea. It was used sporadically in Europe as an antethoracic subcutaneous transplant since 1911. However, very little has been written concerning the intrathoracic replacement of the esophagus with the colon. Robertson, Howe, and Smithwick[17] reported, before the Surgical Forum in 1952, a successfully staged operation in which the transverse colon was interposed between the esophagus and jejunum. Kergin, in 1953,[10] used an isolated transverse colonic segment to bypass a high esophageal obstruction caused by a benign paraffinoma. In 1954, Mahoney and Sherman,[11] and Dale and Sherman[4] reported upon their experiences using the right colon for reconstructing the esophagus in carcinoma and tracheoesophageal fistula with atresia. Recently Haight[7] has successfully used the right colon in 7 cases for delayed reconstruction of the esophagus in tracheoesophageal fistula with atresia.

In order satisfactorily to replace the esophagus, the substitute should meet certain criteria. It should have sufficient length, be assured of continuing viability and the lining be resistant to acid peptic digestion. The colon appears to meet these requirements and in addition offers other distinct advantages. The colon has an excellent blood supply. The anatomic distribution of the marginal arteries is such that a segment of viable bowel suspended upon a pedicle of the middle colic artery and vein can be mobilized from the cecum to the descending colon.

Recent evaluation of the poor results obtained in resection for carcinoma of the esophagus significantly point to the high incidence of local anastomotic recurrence due to inadequate longitudinal removal of the esophageal cancer.[20, 24] The colon is of sufficient length to reach the neck in high esophageal resections. This allows one to resect as much of the esophagus as deemed necessary and do an anastomosis without tension at the proximal suture line. Although the majority of our esophago-colon anastomoses have been performed within the thorax, it may be better to do them in the neck. Less proximal colon has to be used when the bowel is displaced through an anterior mediastinal tunnel into the neck because of relatively short distance between the xiphoid and the manubrium. The vascular pedicle is not angulated or put under tension as is occasionally true when the colon is displaced into the right chest over the right lobe of the liver. One great advantage of doing the anastomosis in the neck is that if leakage occurs the resultant fistula can easily be closed and will not be fatal.

We have been particularly impressed with the length of the colon in children. This is due to the redundancy of the transverse colon and the presence of a thin pliable mesentery. Thus, it is particularly applicable in reconstituting an adequate swallowing mechanism in congenital atresia or corrosive strictures where long segments are involved.

The large bowel is seemingly resistant to peptic ulceration clinically even though reflux of barium into the colonic segment can be demonstrated fluoroscopically, no evidence of colitis could be seen on routine esophagoscopy and post-mortem examination up to 20 months following implantation.

Our experimental evidence supports the above concept. Furthermore, Merendino and his associates[12, 22] subjected dogs to histamine stimulation 30 days after colon transplants. None of their animals exhibited esophagitis or colitis. However, in most of his animals, severe gastritis was present.

The absence of esophagitis clinically and the inability to produce it experimentally when the colon is interposed between the esophagus and stomach suggests its applicability in resections for peptic esophagitis. Although 2 patients had complete amelioration of their symptoms, their follow-up has been too short to propose this operative procedure as a cure in these individuals. With the evidence at the moment, it appears that the interposition of an acid resistant segment of bowel between the esophagus and stomach protects the usual susceptible esophageal muscosa from digestion by regurgitation of gastric juices. Also the use of a segment of bowel between the esophagus and the stomach allows the latter to remain in its normal position below the diaphragm.

Fluoroscopic examinations show regular contractions which propel the bolus along the transplanted colon to the stomach. Thus, the colon serves as a conduit between the esophagus and the stomach. This, along with the stomach remaining in a normal position below the diaphragm, accounts for the excellent nutrition of these patients.

SUMMARY

1. The technique of reconstruction of the esophagus with segments of the colon is outlined.

2. Experimental data are presented to support the thesis that the colon can serve satisfactorily as an esophageal substitute without the development of colitis or esophagitis.

3. The results and complications in 28 patients operated upon for benign and malignant disease are discussed.

4. The advantages of using the colon instead of the stomach or jejunum are presented.

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Discussion

DR EARLE B MAHONEY, Rochester, N Y—Thank you, Dr Haight, for the privilege of discussing this paper Dr Sherman, Dr Dale, and I have been extremely interested in following Dr Neville's work and we are delighted with the results he has had, with his present enthusiasm for the procedure, and the beautiful presentation given today Our series is not as large as Dr Neville's but I think we agree in every way with what he has said We are particularly interested in using this as a procedure for establishing continuity of the gastrointestinal tract in children with benign lesions The original procedures were for carcinoma of the upper esophagus and tracheoesophageal fistulas in which we could not establish normal continuity by direct anastomosis Those children are now growing and are well at 3 years of age We also feel that in the case of carcinoma of the esophagus, as mentioned by Dr Neville, that if one can do a total esophagectomy the end results will be better We have used this several times as a two stage procedure in poor risk patients, resecting the carcinoma as the first stage through a thoracic incision on the right, leaving a cervical esophagostomy and a gastrostomy As soon as the patient's general condition improves the gastrointestinal continuity is restored by a colonic transplant in the anterior mediastinum which does not require a second thoracotomy

DR FRED JARVIS, Seattle, Wash—Although we too have used the colon in replacement of the esophagus, I should like to present a somewhat varied situation, which we encountered recently, with the thought that its solution might be of value to others in the Association

(Slide) This lady, aged 34 years, presented herself with a markedly decompensated achalasia of the esophagus that held 1,300 cc In 1948, we did the then accepted cardioplasty procedure The result was only moderately successful as we had found in a series of 9 cases at that time, and we classed her as a fair result only However, her symptoms continued mildly for approximately 6 years and then more severely for a year because of reflux esophagitis, until she finally was unable to get anything through the strictured lower end of the esophagus, even liquids We then chose to interpose a segment of jejunum about 25 cm in length, this operation having been applied in 2 cases of achalasia at the time, very successfully I believe, by Merendino and Dillard The result again was only moderately and briefly successful, because at the end of 6 weeks she was again totally unable to eat

(Slide) This was the stricture that existed at the time she returned

Six weeks after the interposition of the jejunum we found a situation on esophagoscopy that was misinterpreted as a stricture of the proximal anastomosis of the jejunum We chose then to enter the right chest since we could not get into the proximal stoma to insert a dilator We found a most interesting situation The large sigmoid esophagus had developed a cecumlike dependent pouch beyond the jejunal stoma The anastomosis was widely opened proximally Two fingers could be inserted into the stomach Weight of food in the esophageal pouch converted the proximal stoma into a slitlike aperture that was physiologic rather than organic The problem of dealing with this became one that we puzzled about for a bit Finally, at the suggestion of my associate, Dr Robert Coe, we decided that if we could convert this very large sigmoid pouch into a straight, though aperistaltic, tube to deliver the food into the peristaltic jejunal hopper below, it might work. We therefore made a wide V excision of the excess esophagus from the thoracic inlet down to the cecum and thus recreated a small, relatively normal sized esophagus

The result has been most satisfactory The patient now for the first time, since she was 18 years old, is able to eat steak and anything else she wants as rapidly as anyone

(Slide) Unfortunately, this slide shows quite poorly the final result There is very slight hesitancy of barium at the orifice of the interposed segment Barium passes otherwise normally and rapidly into the stomach

DR GEORGE I THOMAS, Seattle, Wash—As you know, we have been interested in the replacement of the esophagus and portions thereof by the jejunal interposition operation, for approximately 5 years, under Dr K A Merendino's guidance, and I thought it would be of interest to the Association to bring up to date some of our recent data

(Slide) This slide was alluded to by Dr. Neville and represents a comparative study of groups of jejunal and colon replacement preparations. Fourteen dogs in the colon series and 10 dogs in the jejunal series were given large doses of histamine for intense stimulation of acid-pepsin secretion and evaluated either upon their death or upon sacrifice after 45 days. As you can see, both segments of the intestinal tract presented esophagitis grossly and microscopically. The colon and the jejunum essentially had the same negligible degree of pathology after this stimulation occurred. One perforation of the colon is included at the gastrocolic anastomosis and, at the time of autopsy, it contained a small amount of inspissated pus, and we therefore perhaps assumed that it was due to a suture line breakdown. You can see a similarity of ulcerative disease of the stomach and duodenum in both series.

(Slide) This is a recent breakdown of 29 clinical cases. The jejunal segments vary in length from 20 to 37 cm., the majority of which (18) were done for benign peptic esophagitis with stricture. All but 2 of these patients had sliding hiatal hernia. We have performed 3 jejunal interposition operations for cardiospasm, and 3 operations for this entity following several previous procedures, esophagogastrostomy and similar operations. We have performed 2 procedures for extrahepatic portal block with varices, and these patients are doing well. There are 3 miscellaneous lesions, 2 lye strictures, and one iatrogenic (?) stricture. That was a lady who developed a severe peptic esophagitis and stricture following prolonged nasogastric intubation after cholecystectomy. The mortality rate is 10 per cent in the entire series.

(Slide) We think this slide will be of interest, because of the fact that all these patients were severe alcoholics. We believe that alcohol is much like histamine in the experimental preparation and puts the intestinal "esophagus" to severe test. As a matter of fact, 3 of these patients had suffered because of their stricture, and were able to resume their very pleasant pastime following the jejunal procedure. They are very happy with the operation.

DR. OWEN H. WANGENSTEEN, Minneapolis, Minn.—One of the great advantages of having graduate students is that unanticipated dividends accrue therefrom not infrequently and, when these men come from other institutions, they often bring with them ideas concerning operations new to the recipient institution, but practices already in vogue from whence they came. The nature of my discussion will outline such an occurrence.

Many of you in this audience, I know, have worked with various types of gastric tubes for esophageal replacement. A number of you have used the Beck-Jianu gastric fundus. A number of years ago (Surg. Gynec. & Obst. 72: 257, 1941), I reported upon 5 patients in whom I had swung up a jejunal loop attaching the lower end to the gastric fundus, the upper proximal portion being brought up subcutaneously in the midline to the nipple level. This procedure was employed to complete the Torek operation for cancer of the esophagus. There was no mortality although I completed few of the procedures.

It is concerning use of the Mes greater curvature tube (J. Internat. Coll. Surg. 11: 270, 1948) in replacing the esophagus in operations for cancer of the upper thoracic segments of which I wish to speak today. Whereas the Beck-Jianu tube, pedicled at the base of the gastric fundus, does give considerable added length, it does not afford the length which a tube designed from the greater curvature does, if one sacrifices the entire lesser curvature of the stomach. Those of you who have measured the length of the greater curvature of the stomach at operation have had occasion to observe that it is in the range of 55 to 60 cm.; the lesser curvature from the pylorus to the esophagus, on the contrary, measures only usually from 20 to 25 cm. In other words, if one begins fashioning the greater curvature tube by cutting away the lesser curvature, beginning a short distance above the pyloric artery, it will be found that it is not difficult to make a tube about as long as a thread placed along the greater curvature from esophagus to pylorus. As has been suggested, this distance is ordinarily in excess of 50 cm. In making such a tube, I use the ordinary half-length, hysterectomy type of clamp, engaging the stomach along the path of the tube to be designed between two such clamps—the upper row of clamps will come away with the sacrificed lesser curvature. When this division has been completed, I remove the lower clamp and under the tension of stretch, I place a series of interrupted sutures in the mucosa as the next clamp is removed and interrupted mucosal stitches are placed at that level, the surgeon taking pains to see to it that the tube is stretched out as it is being fashioned. When the mucosal suture has been completed to the very top of the gastric fundus, a similar row of interrupted sutures (4-0 silk) is placed in the serosa.

A Heineke-Mikulicz pyloroplasty is done to insure ready gastric emptying. The length of the tube is measured upon the anterior chest wall to see that it will reach well into the neck. It is then brought up through the esophageal hiatus. I do these operations conventionally through the right thorax. A short transverse incision is made in the neck on the right side just above the clavicle. With two fingers of the left hand indenting and elevating the pleura, the neck wound is enlarged until the fingers from within the thorax protrude through the neck wound. The anterior portion of the sternocleidomastoid muscle is cut free from the clavicle, thus enlarging the wound and making it possible to pull the gastric tube through the esophageal hiatus behind the right lung into the neck.

I have done only one such operation. I did it in two stages, bringing the cervical esophagus out just beneath the cricoid cartilage beside the gastric tube. It could, possibly, have been done as a one stage procedure. There seemed to be some question of the viability of the upper end of the gastric tube. However, as it proved, no slough in the gastric tube occurred. A catheter was left in the esophageal end for purposes of removing the swallowed saliva by suction. A four-hole No. 14 F duodenal tube was placed in the retroperitoneal portion of the duodenum by the Witzel technique, its tip being threaded well into the gastric tube which lay within the thorax. During the early postoperative course, suction was applied to this tube to keep the gastric tube empty. A feeding jejunostomy was made also on the efferent loop of the jejunum just beyond the duodenojejunal angle.

Approximately 2 weeks after the primary procedure, union was established between the upper end of the gastric tube and the cervical esophagus by a single row of sutures. Disarticulation of the proximal end of the clavicle made this suture easy. On completion of the procedure the capsule of the medial end of the clavicle was tacked down with a few sutures of interrupted chromic catgut. This patient has done well and has no trouble in swallowing. There was a temporary cervical fistula which closed quickly.

A number of surgeons have expressed deep disappointment and a sense of frustration over their accomplishment with cancer of the upper end of the esophagus. In his Judd Lecture at the Medical School of the University of Minnesota in February of this year, Mr. Philip Allison, distinguished Nuffield Professor of Surgery at Oxford University, England, indicated that he had given up operating for cancer of the upper thoracic esophagus because of the high mortality and the low cure rate. A number of distinguished American surgeons who have had a special interest in surgery of the esophagus also have sounded similar notes of discouragement over surgical removal of the proximal half of the thoracic esophagus for cancer.

(Addendum on proof reading discussion, July 3, 1957.—Since this discussion took place, the writer has twice swung up a Mes greater curvature gastric tube into the neck for cancer of the upper thoracic esophagus. In both instances, a one stage anastomosis was made. Both concerned very difficult cancers, one of which was a frankly palliative procedure, in that the patient has involvement of the right lung and an esophagobronchial fistula between the esophagus and the right main bronchus. It was believed that division of the esophagus above the fixed cancer would mitigate the effects of the esophagobronchial communication. The lower end of the esophagus was anastomosed to a jejunal loop to provide a drainage vent for the esophageal secretions. This patient died a few weeks after operation from complications of the fistula. A small leak developed late in the neck wound, which did not affect the issue. Inasmuch as the cancer had been left in situ, the Mes tube had been brought up behind the sternum. In the other patient, even though the esophagus and trachea seemed to be fused into a single indurated mass, it was possible to separate them, working alternately from above and below. This patient has done well.)

In looking back upon this very limited experience with only 3 cases, the writer feels constrained to remark that the simpler convalescence of the patient with the two-stage procedure was noticeable. It was also the easier cancer to extirpate. At this time the writer feels that an added experience with both the one- and two-stage procedures will be necessary to determine which is the safer and better operation. Certain I am that this operative technique has something to offer in the management of cancer of the thoracic esophagus.

It is my impression that use of the Mes greater curvature gastric tube may go a long way to simplify some of the difficulties of esophagectomy for cancer of the upper half of the thoracic esophagus. Moreover, I believe that its use may also play an important role in improving the end results in that a greater length of the esophagus proximal to the lesion will be sacrificed in the extirpation of the cancer.

DR. RICHARD H. SWEET, Boston, Mass.—I would like to make one or two comments about the use of the colon. This is something we are all very much interested in nowadays, and it has great use. I believe with Dr. Mahoney that its utilization perhaps should be confined to 2 indications: (1) the use for substitution in cases of stricture and other benign lesions, particularly with regard to making an anastomosis in the neck, and (2) in cases of carcinoma for palliation only.

It is much easier to accomplish in children than it is in adults; at least that has been my experience. From the point of view of technique, so far as I am concerned, the exact methods of doing this are not quite established satisfactorily. I would like to point out to you that in cases of technical difficulty, which I shall not go into at the present time, I have found it useful to make a sternotomy incision all the way through the sternum. This can then be extended into the abdomen. This method has many advantages, one of which is that the surgeon does not have a second team that gets in his way.

One other comment is about the function or the physiologic behavior of this colon transplant with which we do not yet have much experience. I have one patient who is enormously satisfied with the operation because she is so much better than she was beforehand, but during defecation she has a most annoying fluttering sensation in her chest.

DR. WILLIAM E. NEVILLE, Cleveland, Ohio (Closing).—I wish to thank all the cussants. Dr. Mahoney was particularly kind. It was due to his initial enthusiasm using e colon as an esophageal substitute, that we embarked upon this program 3 years ago.

Dr. Jarvis mentioned using the jejunum and I believe we can go along with what he said. When we started this project it was our purpose to evaluate the colon only as an esophageal replacement. It may be better in lower esophageal lesions to use the jejunum. It is easier to obtain a segment of jejunum for a short lower esophageal replacement than to mobilize the colon.

We have been interested in following the work of Drs. Thomas and Merendino with regard to the interposition of a jejunal segment between the lower end of the esophagus and the stomach. Their results seem to be good. He did not mention whether they had used the colon clinically, but I know they operated upon a number of experimental animals using the colon and came to the conclusion that it has a certain inherent resistance to acid peptic digestion.

I thank Dr. Wangensteen for his comments. So far as using the stomach in the manner in which he depicted, even though it appears to be relatively easy to do—isn't this what we are trying to get away from in using the colon? With a tube of the stomach being used to replace the esophagus, you may end up with symptoms of esophagitis.

I agree heartily with Dr. Sweet. I disagree a bit on the point of palliation for carcinoma, if we can use that term. As I mentioned, we attempted to bypass 4 nonresectable carcinomas. In none were we ever able to institute roentgen therapy and none left the hospital. He is absolutely right in that it is much easier to mobilize the colon in a child. The colon seems to be longer, it has a thin pliable mesentery and you can get a much longer segment. The sternal incision is certainly an interesting approach. We have not used it because we have had no difficulty in obtaining an adequate anterior mediastinal tunnel.

I thank the Association for the privilege of the floor.

PRIMARY TUMORS OF THE HEART

A SURGICAL PROBLEM

J. GORDON SCANNELL, M.D., AND HERMES C. GRILLO, M.D. (BY INVITATION)
BOSTON, MASS.

PRIMARY tumors of the heart are clearly a surgical problem. As an increasing number of cardiac operations are carried out, a greater number of primary tumors of the heart will be recognized. Since the rapidly advancing techniques of cardiac surgery will certainly permit successful removal of many of these tumors, greater awareness of their clinical behavior and the employment of appropriate methods in their diagnosis are in order.

We wish to report the successful removal of two primary cardiac tumors. The first, a myxoma of the left atrium, was removed 22 months ago. The patient is now well. The second, a fibrosarcoma of the right atrium, was removed 12 months ago. This patient is presently well and without evidence of recurrence. A third patient, with an intra-atrial myxoma, died suddenly while awaiting diagnostic studies which almost certainly would have led to operation.

CASE 1.[30]—The patient was a 33-year-old construction worker. He had been in excellent health until, 18 months before admission, he noted minimal hemoptysis during the course of a severe respiratory infection that was accompanied by an unusual degree of dyspnea. These symptoms subsided, but a second hemoptysis occurred 12 months later and again one month before admission. The dyspnea and wheezing associated with the latest episode were persistent and disabling. He therefore entered the hospital for study. There was no history of rheumatic fever.

On admission, the patient was dyspneic at rest. A few râles were noted at the right lung base, but there was no other evidence of congestive failure. Examination of the heart showed the classic signs of "pure" mitral stenosis. There was slight enlargement. The rhythm was regular. At the cardiac apex there was a presystolic roll of Grade 3 intensity, ending with crescendo in a sharply accentuated first sound. There was an associated presystolic thrill. The pulmonic second sound was moderately loud and there was an opening snap. No systolic murmurs were detected.

Electrocardiogram showed the pattern of right ventricular hypertrophy. X-ray study (Fig. 1) disclosed a slightly enlarged, triangular heart shadow with fullness of the left atrium and congestion of the lung roots.

Mitral commissurotomy was advised and operation carried out on May 23, 1955. The atrial appendage was of normal size and configuration, providing easy entry into the atrium. The latter was found to contain a soft lemon-sized mass attached by a narrow pedicle to the base of the interatrial septum close to the posteromedial commissure of the mitral valve. The tumor had prolapsed into the valve orifice, seriously obstructing blood flow through that structure.

From the General Surgical Services of the Massachusetts General Hospital and Department of Surgery of the Harvard Medical School

Read at the Thirty-seventh Annual Meeting of The American Association for Thoracic Surgery at Chicago, Ill., May 4-7, 1957.

Under the existing circumstances—particularly since we were aware of the disasters which had invariably occurred[16, 24] when impromptu attempts to remove such tumors had been made—it was thought best to postpone removal until, under hypothermia, the left atrium could be opened and the tumor excised deliberately. The first operation was therefore terminated.

A second operation was carried out on June 15. With the patient's body temperature lowered to 26.4° C. (lower than had actually been planned), the venous inflow to the heart

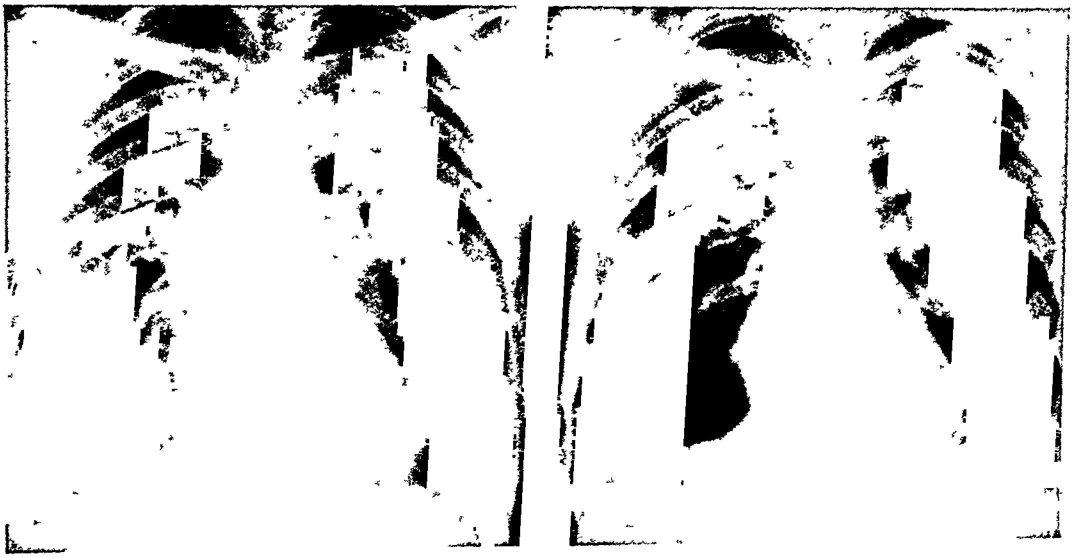

Fig 1.—Case 1 Preoperative and 2 month postoperative posteroanterior roentgenograms. The preoperative film shows a slightly enlarged heart with a straight border and congestion of the lung roots The postoperative film shows a return to normal.

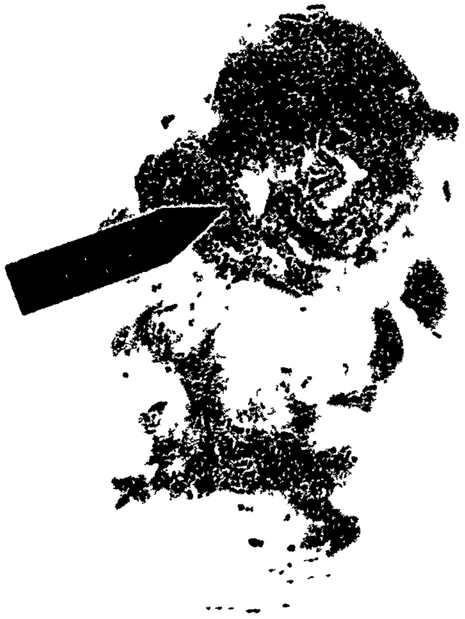

Fig. 2.—Case 1. The arrow indicates the point of attachment of the myxoma to the interatrial septum The inferior pole of the tumor is grooved where it prolapsed through tral valve orifice.

was occluded, the left atrium opened, and the tumor removed (Fig 2) There was total interruption of the circulation for 5½ minutes Ventricular fibrillation occurred immediately upon removal of the tumor Normal rhythm was restored only after 2 hours of cardiac massage and elevation of patient's body temperature to 27 5° C The patient's recovery thereafter was uninterrupted

The patient's postoperative course was comparatively uneventful He was discharged to convalescent care on the twenty fourth day, he returned to work as a steel construction worker 7 months after operation Twenty three months (April, 1957) after his initial operation he was entirely well

The final pathologic report on the tumor removed was myxoma

This patient illustrates very well the problems of an intracardiac myxoma This is the most common primary tumor of the heart, accounting for one third of the reported primary cardiac neoplasms The majority of myxomas occur in the left atrium where they arise from the rim of the fossa ovalis in the interatrial septum They are usually pedunculated and may cause ball valve obstruction of the mitral orifice The result is a clinical picture indistinguishable in most respects from mitral stenosis [1, 24, 26, 33]

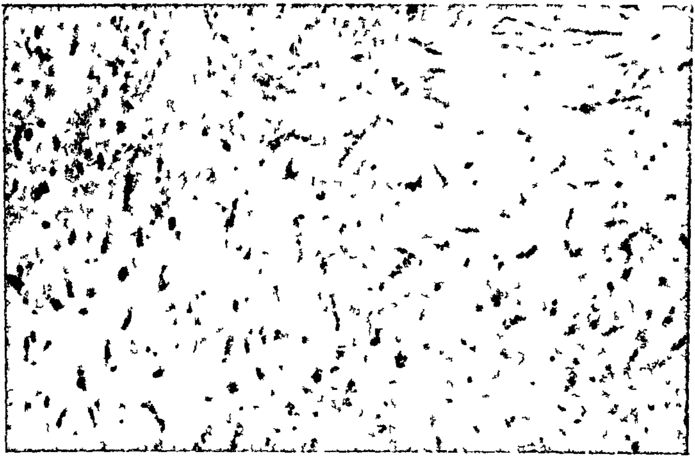

Fig 3 —Case 1 Myxoma One of the typical microscopic fields

Features which suggest intracavitary tumor instead of mitral stenosis are (1) the absence of a history of rheumatic fever, although mitral stenosis without identifiable rheumatic fever in the past is not uncommon, (2) changes in symptoms and murmurs with changes in position, (3) syncope, (4) a rapid progression of symptoms, and (5) marked failure of response to medical therapy [29, 33] None of these is diagnostic

If the tumor occurs in the right atrium, obstruction to the tricuspid valve may occur In this event, one may find the clinical features of tricuspid disease and signs and symptoms of predominantly right-heart failure. As such, they may closely resemble those of constrictive pericarditis as one of Bahnson's cases demonstrates.[2]

The microscopic picture (Fig. 3) shows a relatively acellular structure of loose connective tissue. The cells vary in size and shape. Some question still remains whether cardiac myxomas are true tumors or organized thrombi. The predominant opinion presently favors neoplasm.[23, 29]

A second case of left atrial myxoma is included here to illustrate several points. In the first place, a change in the character of the murmurs and the occurrence of peripheral arterial emboli in the presence of a normal cardiac rhythm led to a presumptive diagnosis of atrial myxoma. Second, death from cerebral embolus occurred, emphasizing the extreme jeopardy in which such patients live.

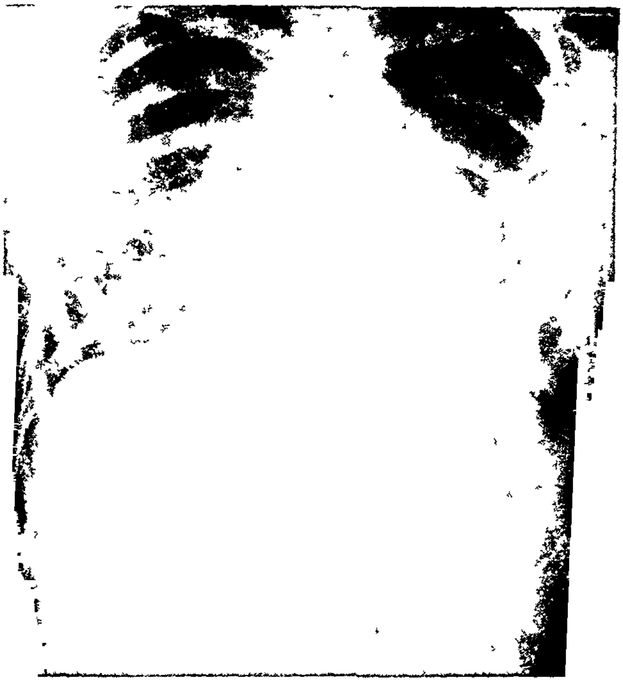

Fig. 4.—Case 2 The film shows cardiac enlargement and congestive changes in the lungs.

Case 2.[11]—The patient was a 9-year-old girl. Eleven months before her final admission, she had had an appendiceal abscess drained. At that time she was found to have an enlarged heart. Two months later, following an interval appendectomy, severe recurrent nosebleeds and low-grade fever led to a provisional diagnosis of rheumatic fever. Salicylates were given and the patient transferred to the House of the Good Samaritan (primarily for children with rheumatic fever).

Three months before admission the patient had a cerebral embolus and an embolus to the index finger of her left hand. There were also several episodes of abdominal pain. Several blood cultures were negative. It was noted that her heart was in regular rhythm. The patient responded poorly to anticoagulant therapy. At this time, atrial tumor was suspected. While arrangements were being completed for angiocardiography in an effort to establish the diagnosis, a second major cerebral embolus occurred from which the patient died (August, 1956).

During the course of the patient's illness the cardiac murmurs changed from a short apical systolic murmur and late diastolic murmur with presystolic accentuation, to a harsh Grade 4 systolic murmur and Grade 3 diastolic rumble Her chest films (Fig 4) over a 7 month period of observation showed considerable enlargement of the heart with congestive changes in the lungs

Autopsy disclosed a gelatinous 5 cm mass (Fig 5) attached to but not invading the left atrial wall The tumor consisted mainly of loose fronds, and tumor emboli were found in the brain, spleen, and kidneys The final diagnosis was myxoma (Fig 6)

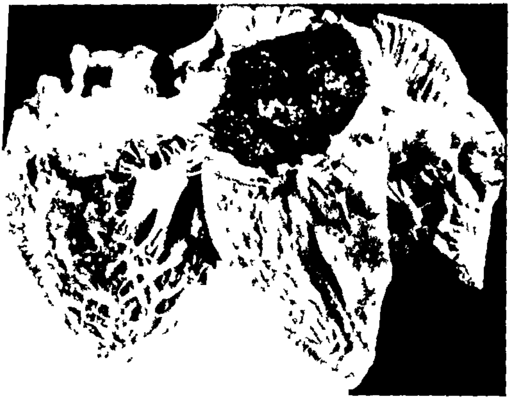

Fig 5—Case 2 The gelatinous myxoma is attached to but not invading the left atrial wall

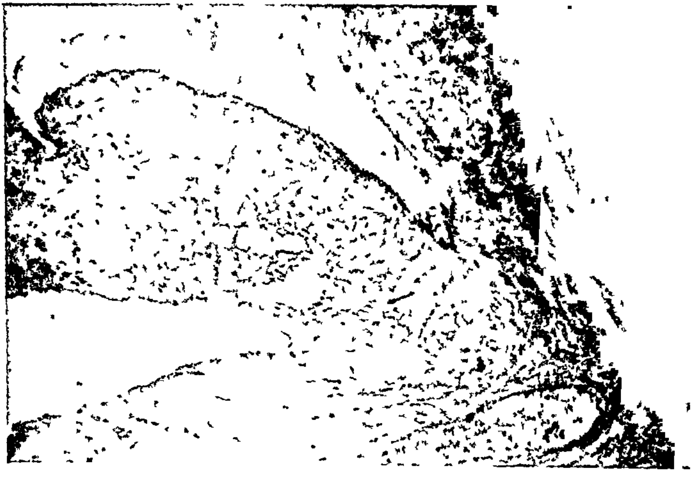

Fig 6—Case 2 Myxoma Microscopic appearance

The occurrence of tumor emboli from auricular myxomas has been frequently described; in fact, in one reported case, the diagnosis of primary heart tumor was made by pathologic study of the removed peripheral embolus.[11]

Since sudden death from embolization, valvular occlusion, or conduction disturbance is common with cardiac neoplasms, diagnosis and treatment are matters of urgency.

Our third case of primary tumor of the heart is that of a low-grade fibrosarcoma of the right atrium. This tumor was excised successfully and the patient is free of disease one year later.

CASE 3.[10]—The patient was a 7-year-old schoolgirl. Her present illness began 4 months before admission with a severe upper respiratory infection, followed by recurrent bouts of fever. During the week before admission, pain and swelling of the epigastrium, anorexia, fatigue, dyspnea, precordial pain, and puffiness of the face were noted.

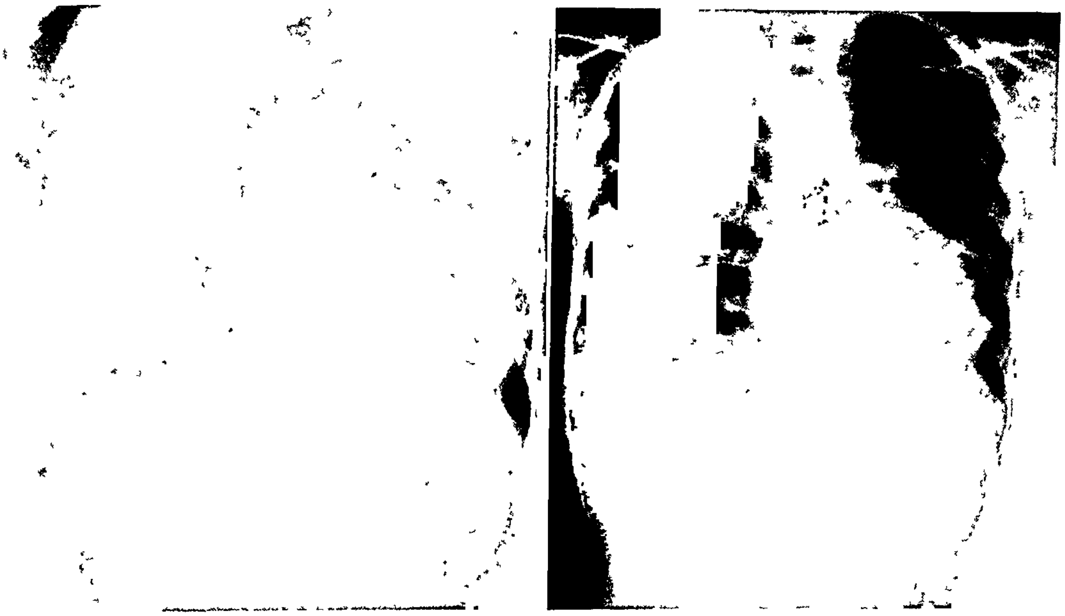

FIG. 7.—Case 3. Posteroanterior roentgenogram before and after pericardiocentesis.

Physical examination, confirmed by x-ray studies (Fig. 7) on admission, was consistent with a sizeable pericardial effusion. On the second hospital day, a pericardiocentesis yielded 400 c.c. of frankly bloody fluid. Following this procedure the patient's clinical state was greatly improved. Chest film (Fig. 7) showed the heart shadow to be almost normal. During the ensuing 2 weeks, x-ray films demonstrated gradual reaccumulation of the pericardial fluid with increasing signs of pericardial tamponade. On the twenty-first hospital day, a second pericardial tap was done with the removal of 400 c.c. of bloody fluid. Cell block and cytologic examination of the fluid were reported negative for tumor, although atypical cells were noted. Clinical improvement again followed pericardial tap, but in the course of the next 10 days, signs of reaccumulation of fluid were apparent and the patient therefore was explored (March 8, 1956) with the purpose of establishing the diagnosis and relieving the tamponade by pericardial-pleural drainage or partial pericardiectomy, if need be.

The pericardial sac was found to be distended by a moderate amount of frankly bloody fluid. As the exploring finger was passed over the anterior surface of the heart, a firm, nodular mass about the size of a walnut was encountered in the region of the right atrial appendage. To visualize this area better, the thoracotomy incision was extended across the sternum. The mass involved the wall of the atrium in the cleft between the appendage and the atrial wall. It was intramural, partly projecting outside the heart, and partly into the right atrial cavity. It was possible to free the tumor sufficiently to apply curved Potts clamps so as to excise the tumor completely with a wedge of atrial wall (Fig. 8). This could be done without interruption of the circulation. The resulting defect in the atrial wall was closed with fine silk, using a flap of atrial appendage to close the gap. The child tolerated the procedure extremely well.

The patient's immediate postoperative course was satisfactory and she was discharged home on the thirteenth hospital day. She re-entered the hospital 2 weeks later with an increasingly rapid pulse and increased venous pressure suggesting recurrent tamponade. The cardiac shadow by x-ray was not particularly enlarged. Under the circumstances, open exploration rather than needle aspiration was considered justified. The operative findings were puzzling in that there was remarkably little pericardial fluid. The heart itself seemed to be somewhat enlarged. There was no evidence of recurrent disease. A large "window" was left in the pericardial sac.

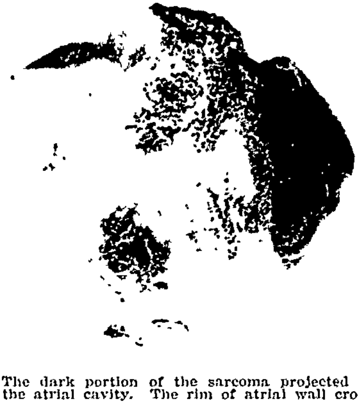

Fig. 8.—Case 3. The dark portion of the sarcoma projected into the pericardial sac, the lighter portion into the atrial cavity. The rim of atrial wall crosses the tumor vertically in this photograph.

The patient's recovery from the second operation was uneventful. One year later (April, 1957), she was reported well without evidence of disease.

A preliminary pathologic report of rhabdomyoma was finally changed to "low-grade fibrosarcoma."

Primary sarcomas of the heart are as variable in histologic types as sarcomas elsewhere. Myxosarcoma, leiomyosarcoma, fibrosarcoma, angiosarcoma, rhabdomyosarcoma and lymphosarcoma have been reported.[23] Taken as a group they are the second most common primary tumors of the heart, forming with myxomas the greatest part of all primary cardiac tumors. Sarcomas may occur anywhere in the heart but are most common on the right side and particularly in the right atrium. Hemopericardium is common. Primary malignancy as well as secondary malignancy should therefore be considered in the presence of grossly bloody pericardial effusion. Sarcomas are intramural, usually replacing the entire thickness of the heart wall and protruding on one or both surfaces.[29, 32]

This patient did not have any of the commonly described disturbances in heart rhythm nor was the tumor large enough to distort the x-ray outline of the heart.

The tumor was microscopically a low-grade fibrosarcoma (Fig. 9). The cellularity varied in amount and type but most of the cells appeared to be mature fibroblasts. The tumor invaded the atrial muscle in the periphery.

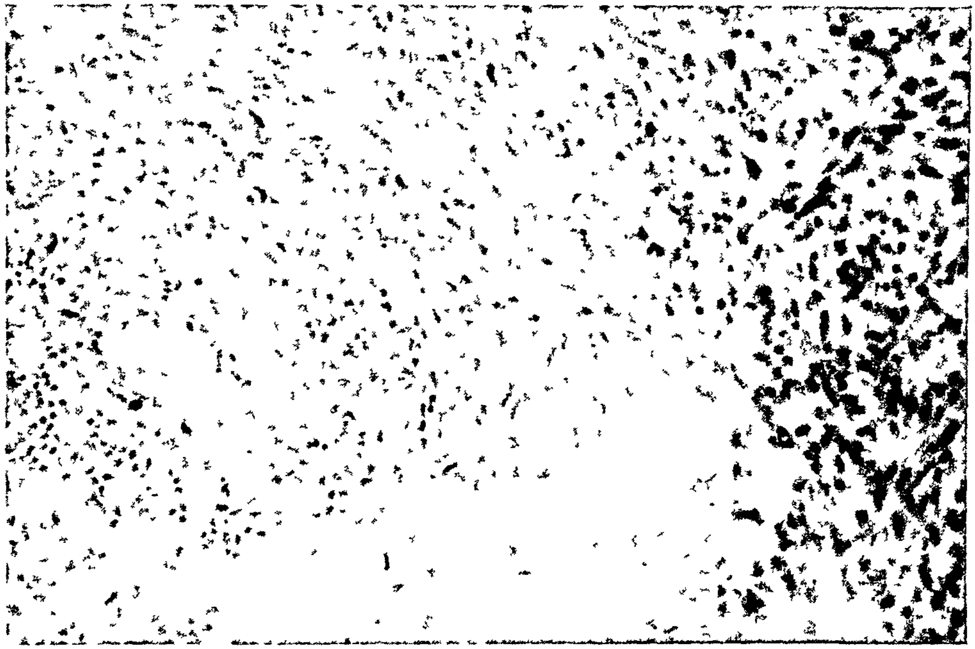

Fig 9.—Case 3. Fibrosarcoma. Microscopic appearance.

DISCUSSION

Cardiac tumors have been extensively reported in the world literature. The three most useful reviews of the subject are by Yater,[33] in 1931, a comprehensive monograph by Mahaim,[26] in 1945, listing a bibliography of 1,298 references, and an admirable clinicopathologic review by Pritchard,[29] in 1951. Wharton[32] reviewed primary malignant tumors of the heart in 1949. Most of the cardiac tumors reported in the literature are secondary, however. These metastatic lesions characteristically manifest themselves by the appearance of cardiac irregularities in patients with known primary malignancy elsewhere and often widespread metastases.[17] The differential diagnosis from primary cardiac neoplasms is readily made.

As Steinberg and his associates[20, 21, 31] have shown, angiocardiography offers a precise method of visualizing intracardiac tumors on the basis of which careful preoperative planning is possible. Beck[5] demonstrated this 15 years ago in the case of an intrapericardial, though extracavitary, teratoma, which he resected successfully. At that time he suggested that atrial myxomas might be removed, either by a closed suction method or by open cardiotomy employing a bypass technique.

A number of attempts, many of them successful, to remove intrapericardial tumors and cysts arising from the surface of the heart have been reported. Gebauer,[19] in 1943, attempted to remove an intrapericardial teratoma. D'Abreu,[1] in 1950, removed a hydatid cyst from the wall of the left ventricle. Hochberg and Robinson[22] in the same year excised a hemangioma of the pericardium involving the myocardium. Maurer,[2] in 1952, successfully removed a lobulated intrapericardial lipoma arising from the left ventricular surface. Longino and Meeker,[25] in 1953, partially removed a sarcoma which arose from the right side of the heart and filled the pericardium but the patient died. Bickford, Egan, and Bryce[6] (1956) report the removal of a mesenchymal tumor from the ventricular surface, and Dabbs, Peirce, and Rawson[18] (1957) the successful excision of a large intrapericardial teratoma.

The first successful removal of an intra-atrial myxoma was carried out by Crafoord,[13, 14] in 1954, using extracorporeal circulation and carefully compressing the mitral orifice and the left ventricle while the atrium was widely opened. In 1955, Bigelow[8] encountered a large left atrial tumor during exploration for mitral stenosis. Rather than postpone removal of the tumor to a later operation, as we elected to do, he proceeded to lower the patient's body temperature by flooding the pleural cavity with cool saline. With the cardiac inflow and outflow clamped, it was then possible to remove the tumor through the open atrium. His patient made an excellent recovery. Our patient represents the third successful removal of an intra-atrial myxoma.

Prior to the cases noted above there have been many attempts to remove intracavitary tumors but without final success. Bahnson and Newman[1] nearly succeeded, in 1953, when they removed the major portion of a right atrial myxoma only to have the patient die 3 weeks later. Death was ascribable in part to residual tumor. The other unsuccessful attempts, many of them almost successful, are included among the references cited.[3, 4, 9, 12, 16, 20, 21-24, 28]

Diagnosis of primary cardiac neoplasms will be made preoperatively in direct proportion to our awareness of their behavior. The knowledge that surgical cure is possible should increase the energy with which these tumors are sought. Once the diagnosis is established, some method of open operation is needed. Almost certainly the use of an extracorporeal pump oxygenator will be the method of approach in the near future, although hypothermia is not without its value. Blind and hurried attempts to remove such tumors are not to be advocated, although it must be remembered that postponement is not without hazard once the diagnosis has been made.

SUMMARY

Two successful excisions of primary tumors of the heart are presented. One, a myxoma of the left auricle, was removed by cardiotomy under hypothermia. The other, a fibrosarcoma of the right atrium, was removed by partial

auricular excision. A third patient, with a left atrial myxoma, died while awaiting studies. The characteristics and treatment of primary cardiac tumors are discussed.

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DISCUSSION

DR. EGBERT H. FELL, Chicago, Ill.—I wish to congratulate Drs. Scannell and Grillo on this very excellent presentation and on their results. Four years ago we had two attempts at one tumor of the heart with unhappy results that taught us considerable. Thus, the reason for this discussion.

(slide) A 16-year-old boy was sent to us from down state with a diagnosis of possible rheumatic heart disease. Fluoroscopy disclosed a large, partially calcified mass in a very large right atrium which swung back and forth into the entrance of the right ventricle and back to the level of the inferior vena cava. Under hypothermia, we planned to close off the inflow tracts but the inferior vena cava was so short and large that we believed it inadvisable. The examining finger in the right atrium felt a pedunculated tumor about 3 inches long, which bobbed back and forth into the right ventricle and back into the inferior vena cava. It originated on the interatrial wall close to the tricuspid valve. The stalk was smooth and approximately 1 cm. in diameter at its base. The distal end of the tumor was 5 cm. in diameter and of firm consistency. We considered it would be a very easy thing to bring it into a blind pouch. The right ventricular pressure was too high to use a well. A small sterile rubber surgical glove was sutured to the right atrium. As we were opening the atrium the heart stopped and we retreated. Circulation was established by cardiac compression and the blood pressure was maintained for 3 days with Levophed. Six months later we went back in, put in another pouch and the examining finger revealed the tumor to have grown considerably in size, it was very friable and portions separated off the original tumor, causing pulmonary emboli.

(slide) The slide shows the tumor close to the tricuspid valve on the atrial septal side, and it is hanging down into the right ventricle.

(slide) This slide shows some of the friable material broken off.

(slide) This photograph shows the pulmonary artery and lung with tumor in the pulmonary vascular tree, causing the sudden exitus on the table.

In retrospect, we should have made a further attempt to remove the tumor at the first operation. It was not friable and could have been removed then. The second mistake was that we waited 6 months to perform the second operation, however, we did believe there were plenty of reasons for waiting.

DR. WILLIAM A. HOPKINS, Atlanta, Ga.—Dr. Fell has just reported a tumor exactly like the one we want to show. The movement of this tumor was so fantastic by fluoroscopy that we obtained a cinefluorographic picture of this which I shall show you.

This was the case of a 26-year-old man who had symptoms of recurring dyspnea over several years, he had also a persistent tachycardia. As soon as he was examined by x-ray the diagnosis was obvious that he had a calcified tumor within the right auricle. Its location was exactly the same as the one presented before. I should like to show the film.

(film) You can see the movement of this calcified tumor. The lower projection of the tumor as it prolapsed into the right ventricle is not shown as well in this film as in the next section. This patient was prepared and operated on under hypothermia and the tumor was removed successfully. He recovered without complication. As you can see, the movement within the heart is fantastic. This man had a persistent tachycardia, the rate ranging from 110 to 120, and after surgery the rate returned to a normal level. One could feel this tumor project forcefully against the right auricular wall, in diastole it would prolapse into the valvular area of the right ventricle.

DR. ROLLINS HANLON, St. Louis, Mo.—I would like to compliment the essayists on their fine results in these two cases and to present one patient who illustrates several points which I think are relevant.

(slide) Regarding the pathologic aspect of these "tumors," they are, of course, tumors in the sense that they are masses within the heart, but it is my impression that many are not really neoplasms but simply organized clots. This is contrary to the prevailing opinion of pathologists as Dr. Grillo has mentioned. This slide shows a higher magnification of a lesion very similar to the one which he showed as a myxoma. This appears to us to be a thrombus, peculiarly organized because of the forces acting on it in the heart.

(slide) This demonstrates the removal of a mass from the right atrium of a 63-year-old woman with signs of cardiac failure, unaccompanied by cardiac enlargement. Five years after onset of symptoms, the diagnosis was finally established by a variety of studies including angiocardiography. She was intensely cyanotic because the mass blocked the orifice of the tricuspid valve, causing a right-to-left shunt through an atrial septal defect. For several months before operation she had extremely low platelet counts with severe hemarthroses and other hemorrhagic manifestations. The relation of the thrombocytopenia to the "tumor" is of interest; we consider it possible that the intracardiac mass was built up by the agglomeration of platelets and other blood elements.

The mass was removed by open atriotomy with the use of the DeWall pump-oxygenator as shown in this movie, cutting away its attachment to the rim of the inferior vena cava and closing the septal defect with interrupted sutures. The patient has done well in the 6 months since operation with prompt and sustained return of platelets to normal levels.

DR. EDGAR P. MANNIX, JR., Manhasset, N. Y.—This very excellent paper by Drs. Scannell and Grillo emphasizes the importance of establishing an early diagnosis in primary tumors of the heart, inasmuch as they may now be successfully removed surgically. It might be of interest to recall that up to 1955 only 13 in some 350 or more cases of primary tumor of the heart had been diagnosed prior to death. We recently had occasion to see a rather unusual case in a 42-year-old nurse who, for 14 months prior to death, had had a history of dyspnea and progressive heart failure without any previous etiological stigma of heart disease. She subsequently developed cyanosis, clubbing of the fingers and toes and polycythemia, and one month prior to death had a cerebrovascular accident. I saw her for the first time 2 weeks prior to death, at which time she had an enlarged right ventricle by x-ray, right ventricular hypertrophy in the electrocardiogram, and an absent pulmonic second sound. She was in intractable right heart failure. On the basis of these clinical data we assumed that she had either tetralogy of Fallot or pulmonary stenosis with an atrial septal defect. I was unable to obtain an angiocardiogram because of her poor condition. She died without opertion. Autopsy was performed.

(slide) This is the right ventricle greatly hypertrophied and dilated. Obstructing the outflow tract of the right ventricle, in the area of the pulmonary valve, is a huge rhabdomyosarcoma. This lesion was independently diagnosed by three different pathologists as being a rhabdomyosarcoma.

(slide) Here you see a close-up view of this tumor blocking the pulmonary valve, and the little slit on the side is the only point of egress for the blood to get out of the right ventricle into the pulmonary artery.

(slide) This shows a small atrial septal defect, which I think explains why she had cyanosis. We believe that as the pressure rose in the right atrium secondary to right ventricular failure, the patient developed a right-to-left shunt and that the cyanosis was due to this defect plus, of course, the stagnant hypoxia secondary to right heart failure.

Inasmuch as, at autopsy, there was no evidence of metastasis of this lesion we believe that had the diagnosis been established fairly early something constructive might have been done surgically for this patient.

DR L FATTI, Johannesburg, South Africa—Thank you for allowing me to speak before you I should like to report one tumor from the heart of a patient, without using either hypothermia or an artificial pump

The patient was a woman aged 45 who showed all the signs of mitral stenosis Many times differentiation is made between mitral stenosis and myxoma, but I do not think any of the diagnostic points are sufficient to make a firm diagnosis

At operation the tumor was easily felt and it was evident that the pedicle was a fairly small one It was a soft gelatinous mass, and the slightest movement threatened to make it move on in the blood stream It was obvious that the only way to prevent it floating off would be to put a finger into the left ventricle beyond the tumor A purse string suture was therefore put into the apex of the heart, a small incision was made and dilated with Hegar's dilators until the index finger of the other hand could be put into the ventricle

The point here is that the tumor was as large as the mitral orifice, which was twice the size of my index finger When the pedicle had been cut through with the nail of the atrial finger, it was quite easy to push the tumor from the left ventricle into the left atrium, and as the atrial finger was withdrawn the tumor was pushed into the auricular hole I had to enlarge the hole in the left atrium a little, give a push and the tumor popped out In fact it popped out so suddenly that we lost about 20 c c of blood My assistant clamped and closed the hole in the left auricle I removed the finger from the left ventricle and pulled the purse string tight

I think it is a diagnostic point of the nature of the myxoma that it should be so easily cut off from the septum, it is an event that has taken surgeons unaware But, even if a Brock's knife is used to detach the tumor, the danger of embolus is the same By this easy method, such disaster is avoided and the operation need not be abandoned I am told by Logan that he applies a knife to the mitral valve by the same ventricular approach

DR ROSS ROBERTSON, Vancouver, B C—The paper of Dr Scannell and Dr Grillo on primary tumors of the heart interests me intensely I presented a paper, similarly titled, before the Pacific Coast Surgical Society in February of this year It will appear in an early issue of the American Journal of Surgery

Five primary cardiac tumors, treated surgically, were reported The pathologic diagnoses were teratoma, cylindroma, mesothelial sarcoma, lymphangioma, and myxoma Two of the patients are apparently cured, one was improved for several years, and 2 died postoperatively The 2 survivors apply directly to this discussion

(slide) The first was a 10 year old boy who complained of dyspnea on exertion Cardiac x rays showed a rounded area of density with calcification in the anterior mediastinum, deeply indenting the right atrium and displacing the pulmonary artery and aorta, as in the cases described by Dr Dabbs yesterday

(slide) Removal of the tumor was difficult because of its close attachment to the aorta It received a small branch from the right coronary artery Pathologic diagnosis Cystic lymphangioma

(slide) This patient was explored for mitral stenosis A myxoma was found in the left auricle The mitral valve was normal The wound was closed and a second operation was planned In retrospect, there were some elements in the history, as described by Dr Grillo, which pointed to the correct diagnosis

(slide) At the second operation, under hypothermia, the venae cavae were taped, the descending aorta was clamped and the coronary arteries were perfused with oxygenated blood through a cannula in the internal mammary artery, a method similar to that of Dr Maloney The tumor, the size of a golf ball, was removed in two pieces It was attached to the auricular septum No residual tumor could be seen

(slide) The patient has made an excellent recovery, as shown in this postoperative chest film (slide) Routine sections showed myxomatous tissue containing many hemorrhagic areas.

(slide) Because the hemorrhagic areas simulated to some extent the early organization of the thrombus, our pathologist examined a number of thrombi, both human and experimental, for comparison. In sections stained with colloidal iron, the tumor was strongly positive for acid mucopolysaccharides, as shown here. The controls were negative.

DR. J. GORDON SCANNELL, Boston, Mass. (Closing).—I should like to thank very much those who have discussed the paper. I must say that the cineradiographic pictures of an intracardiac tumor in action are most dramatic and not a little alarming. The pathologic considerations that Dr. Hanlon has brought up are very interesting. We have discussed the matter at length with the members of our department of pathology and have accepted their considered opinion that these probably represent tumors although certainly the idea that they may represent organized thrombi is well rooted in the literature. As you know, many of these were originally called polyps of the heart.

In closing, I would like to emphasize one point in particular. In most cases of myxoma of the heart, the diagnosis is going to be made in the course of an operation for mitral stenosis. Ideally, the surgeon should proceed at this time with open operation and removal of the tumor. If he is not in a position to do this, whether by cardiac bypass or hypothermia, then it is our conviction that it would be better to postpone final attack upon the tumor to another day. Admittedly, this is a calculated risk, but at the present stage of affairs, we believe it is a justified one.

VENTRICULOPLASTY FOR CARDIAC ANEURYSM

Charles P. Bailey, M.D., Houck E. Bolton, M.D. (by invitation),
Henry Nichols, M.D. (by invitation), and Richard A. Gilman,
M.D. (by invitation)
Philadelphia, Pa.

CARDIAC aneurysm usually involves the left ventricle and commonly is associated with certain physiologic and clinical features which bode ill with respect to the patient's welfare and longevity. Etiologically ventricular aneurysms are of three types: (1) those due to congenital malformation—often described under the term "diverticulum," (2) those resulting from trauma (nowadays usually produced by cardiac surgery), in which case the sac usually is formed by certain extracardiac structures, especially the pericardium (false aneurysm), and (3) those resulting from weakening and bulging of a relatively localized area of myomalacia of the heart wall. These latter most often result from acute myocardial infarction. However, localized forms of syphilis,[1, 2] tuberculous caseation,[3] nonspecific mediastinal abscess,[4] and even the anomalous origin of a coronary artery from the pulmonary artery[5] have been indicted as causative of ventricular aneurysm.

The actual incidence of aneurysm formation following myocardial infarction is subject to considerable dispute, various authors placing it as low as 5 per cent,[6] while others consider the rate of development of this complication to be of the order of 20 per cent.[7] The world literature generally establishes it at about 10 per cent.[8, 9, 10] Part of this controversy devolves about the criteria of definition, some[5, 11] considering that any enlargement of the ventricular lumen due to a localized encroachment upon the thickness of the ventricular wall constitutes an aneurysm. Others restrict the term to those grosser deformations of the heart which produce an obvious outward bulging of the ventricular outline (Fig. 1). Naturally these latter lesions are characterized by a more severe physiologic abnormality and by a more rapidly progressive downward clinical course.

Whether the incidence finally be established at 5 or 20 per cent, the high current rate of occurrence of acute myocardial infarction which is estimated variously by Master[12] at 500,000 to 800,000 per year in the United States suggests that between 25,000 and 160,000 new cases of ventricular aneurysm develop annually in this one country. Since the study of Schlichter, Hellerstein, and Katz[5] places the average life span (with invalidism) following the appearance of aneurysm at somewhat over 2 years, there should be at least twice as many of these patients alive at any given moment. The miserable and progressively downhill course which most of them manifest with conservative

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management suggests that every effort should be made to improve the medical and surgical armamentarium available for the treatment of this disease. It is the purpose of the authors to show that an effective and generally satisfactory method of surgical attack for this condition now exists. To date, 9 patients with ventricular aneurysm have been operated by us, and 8 have survived in a much improved condition.

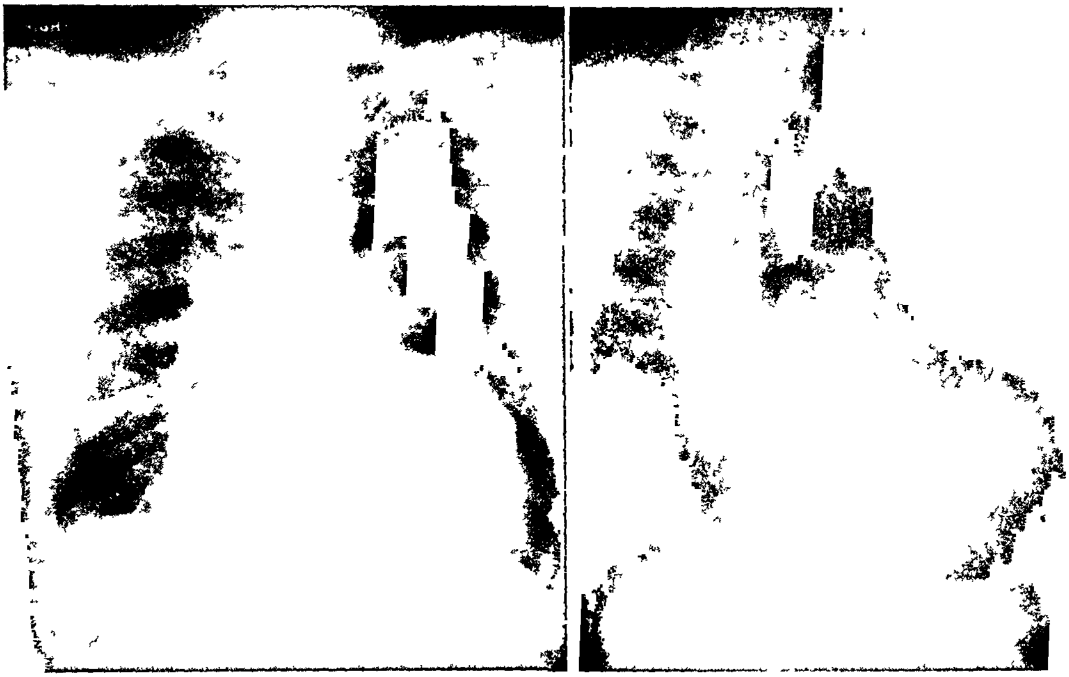


Fig 1.—PA and oblique roentgenograms obtained from J. L., a patient with huge indisputable left ventricular (postinfarctional) aneurysm

CONGENITAL DIVERTICULUM OF THE VENTRICLE

This condition would seem to qualify by all logical criteria as a ventricular aneurysm. It exists at birth as a muscular outpouching usually of the left, but occasionally of the right ventricle, or even of both. Since there usually is free luminal communication with the indicated ventricle the latter type of lesion (open to both ventricles) may permit a physiologic shunt of blood at this level. Other than this relatively rare communicating lesion there characteristically is no physiologic abnormality associated with a congenital ventricular diverticulum since the muscular walls are continuous with and contract synchronously with the ventricles

The chief danger in these cases is that the abnormal muscular outpouching eventually undergoes rupture with resulting fatality. The total (although limited) world experience with this uncommon lesion indicates that it is best treated by surgical excision at an early age.[13-20] An illustrative report of a personal case follows.

CASE 1.—K. W. (Hospital No. 54 01397), a 7-day-old white female infant, was admitted to the pediatric service of the Hahnemann Hospital in Philadelphia on Feb. 4, 1954. Her complaints were those of a feeding difficulty but it had been noticed that there was an abnormal, obliquely directed mass in the epigastric region which pulsated synchronously with the cardiac contractions (Fig. 2). The overlying skin was thin and abnormal, suggesting a central defect of union of the bodily segments. Angiocardiography showed a communication between the left ventricle and an abnormal blood-containing chamber. The diagnosis of congenital diverticulum of the ventricle was made and operation was carried out on Feb. 11, 1954. The mass was dissected free of the overlying abnormal skin and adjacent fibromuscular tissues. It was found to be covered with an extension of the pericardial sac and to be directly continuous with the apex of the heart. There was no opening into either the peritoneal or pleural cavities. The apparent site of junction of the mass with the ventricle was cross-clamped with a curved pair of Potts multiple-toothed forceps. The mass continued visibly to contract in synchronism with the ventricles.

The diverticulum was amputated along a line 5 mm. distal to the clamp (Fig. 3) and the cut end was oversewn using one row of continuous mattress sutures of 3-0 cotton with a swedged-on curved needle. Free luminal communication was demonstrable. A second running suture was used to complete the repair. The clamp was removed and the abdominal wall was closed anatomically.

The postoperative course was uneventful and the infant was discharged from the hospital in good condition.

TRAUMATIC ANEURYSM OF THE VENTRICLE

Accidental injuries, particularly those of a gunshot or stab wound type, may be counted upon to create an occasional aneurysm of the ventricular wall. However, the rapidly increasing incidence of surgical ventriculotomies (with a certain percentage of poor healing) promises a considerably larger harvest of ventricular aneurysms of traumatic origin in the near future. Probably only the fact that most ventriculotomies are made into the relatively low tensioned right ventricle prevents the rate of aneurysm development from being strikingly high. Brock[21] has mentioned this as a complication of pulmonary valvulotomy and infundibulectomy. Indubitably, meticulous care in the performance of the ventricular repair will tend to reduce the incidence of the complication. This is especially pertinent with respect to the larger ventriculotomies such as are employed in current "open heart" techniques.

Undoubtedly, the first step in the mechanism of development of such an aneurysm is a partial dehiscence of the deeper layers of the myocardial line of closure. In many instances, this may be due to failure to appose accurately the inner aspect of the lips of the incision, perhaps due to failure to apply the sutures deeply enough. Thus, a site for beginning dissection of the wound surfaces is provided. This mechanism would seem to be the determinative cause in both cases of traumatic aneurysm which are cited in this presentation. In each, a relatively small left ventricular wound was closed rapidly with surface approximating sutures, the smallness of the ventricular opening preventing certainty of inclusion of endocardial tissue within each of the individual sutures.

Other factors, more difficult to assay, include metabolic deficiencies, [illegible] tension within the ventricular chamber, hypoproteinemia, anemia, and [illegible] specific and nonspecific degenerative diseases of the myocardium.

It would seem probable that a beginning dissection of the ea[illegible] would require at least several days for completion, thus enabling [illegible]

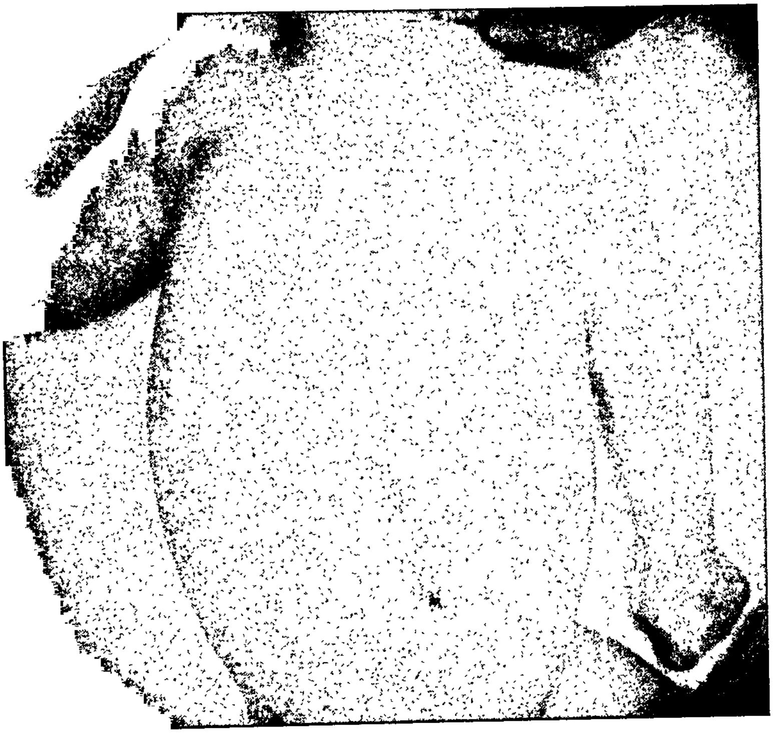


Fig. 2.—Photograph of infant, K. W. (Case 1), showing obliquely directed pulsating epigastric mass and abnormal line of skin fusion. (Bailey, Surgery of the Heart, ed. 1, Philadelphia, 1955, Lea & Febiger, p. 408.)

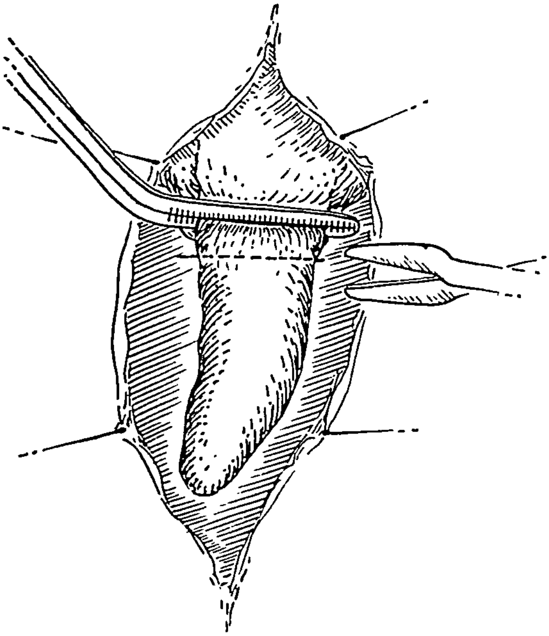


Fig. 3.—Line of indicated section (dotted). (Bailey, Surgery of the Heart, ed. 1, Philadelphia, 1955, Lea & Febiger, p. 411.)

adhesions to develop between the surface of the heart and the overlying pericardium (when it has been reconstructed) or other contiguous juxtacardiac surface (mediastinal tissues, sternum, lung, etc.) Further dissection and stretching of these apposed adherent tissues will serve to create a sizable aneurysmal sac of false type. In most instances, this will communicate with the left ventricular lumen by a relatively narrow transmural channel. Sometimes the communication may be multiple, as in the first of our personal cases (Case 2) Whether any or much thrombosis will develop within such a false aneurysmal sac depends upon the size of the junctional passage and upon the freedom of blood ingress and egress during each cardiac cycle. In Case 2, as soon as the exploring finger tip was applied in such a way as to block blood flow through the larger of the two luminal communications, thus greatly reducing the amount of ebb-and-flow, the liquid blood content of the sac promptly became coagulated (within 4 minutes).

Such false aneurysms of traumatic origin may be presumed to superimpose some additional physiologic burden upon the left ventricle, since the semi-elastic aneurysm visibly becomes distended during systole and tends to collapse partially during diastole. In other words, a certain amount of paradoxical function is imposed upon the left ventricle thus reducing the efficiency of its gross stroke output. However, because of the usual small size of the communicating transmural passage the effective loss of output characteristically is not great and can be borne for a considerable period of time. However, one of our 2 patients (E S, Case 3) exhibited signs and symptoms of serious lowering of myocardial reserve at the time of secondary surgery.

Should massive or obliterative thrombosis reduce the aneurysmal lumen the effect would be to diminish the amount of the paradox and thus to lessen the physiologic burden Complete cure by such a mechanism is not inconceivable although presumably it is improbable The likelihood of arterial embolization, due to extension of the thrombotic process into the ventricular lumen or to detachment of a loose fragment from the sac, would seem to be small in these cases again because of the usual narrowness of the transmural channel

Indeed the greatest hazards to the patient with traumatic (false) aneurysm of the ventricle would seem to be the twin probabilities of continued growth of the sac (causing pressure symptoms) and its tendency to eventual rupture The likelihood and imminence of such developments would seem to be dependent upon the tightness of the restricting adhesions, the tensile strength of the tissues making up the false sac, the size of the ventricular communication, and the level of intraventricular pressure during systole[22]

CASE 2—G F. (Hospital No. 55-10624), a 31 year-old white housewife was admitted to the Hahnemann Hospital in Philadelphia on Sept 16, 1955, for investigation of an abnormal shadow evident upon the left cardiac border This had developed shortly after a mitral commissurotomy had been attempted at another clinic 4 months previously. On that occasion (May 8, 1955), the wall of the left ventricle inadvertently had been perforated from within by the index finger of the operating surgeon. This had resulted in massive blood loss and the onset of ventricular fibrillation. The attempt at commissurotomy was terminated promptly, hasty suture repair of the laceration was carried out, and the heart was defibrillated successfully with electric countershock.

The postoperative course was stormy but finally the patient was discharged in good condition with clinical relief of the symptoms caused by the initial obstruction. However, in August, 1955, an abnormal bulging shadow was observed along the left cardiac border (Fig. 4, *A* and *B*). In view of the history it was suspected that this might be a ventricular aneurysm. During the next few weeks the shadow was seen radiographically to increase in size, and the patient began to complain of precordial discomfort.

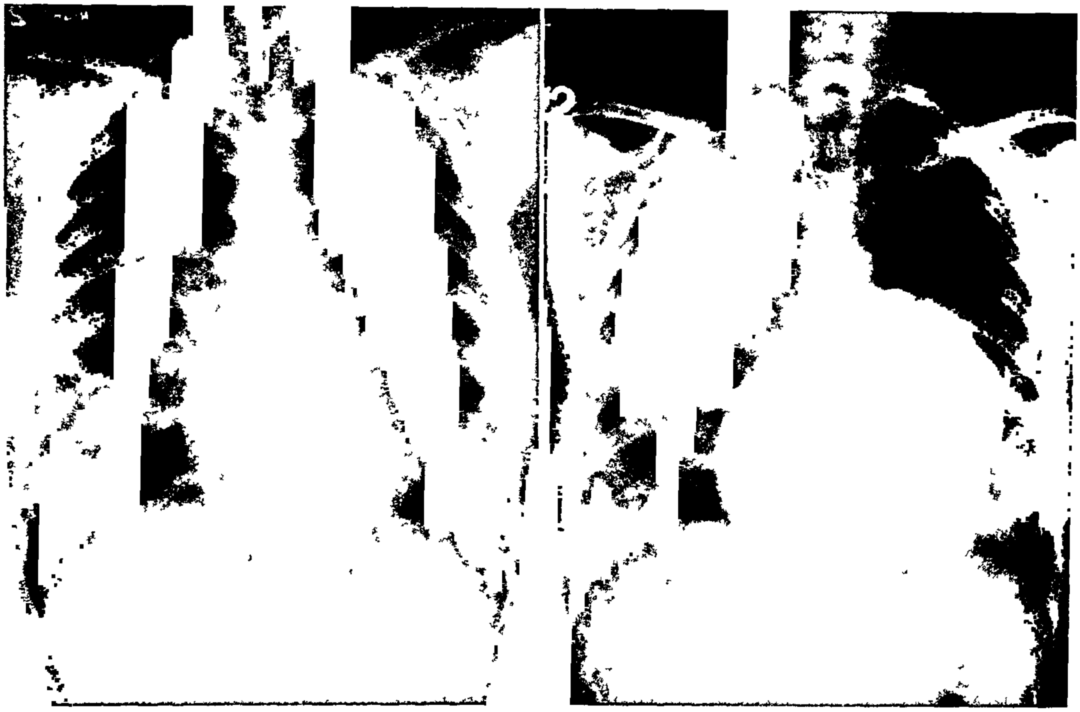


Fig 4.—*A*, PA roentgenogram of G. F. (Case 2) taken prior to first commissurotomy attempt.

B, PA roentgenogram of G F. taken a few weeks after inadvertent laceration of the left ventricle The abnormal shadow was suspected of being a ventricular aneurysm.

(Smith and associates, Surgery 42: 497, 1957)

The patient had a normal sinus rhythm, a sharp first sound at the apex, and a mid late rumbling diastolic murmur. The pulmonary second sound was accentuated.

The electrocardiogram showed an elevation of the S T segment in Leads I, AVR, and AVL. There was S T segment depression in Leads II, III, AVF, and V_4 through V_6. The T wave was inverted in Leads I and AVL. The tracings were considered suggestive of damage to the high lateral wall of the left ventricle.

The x-ray investigation included kymographic studies which indicated pulsations in the mass of 10 mm. amplitude and these were paradoxical to ventricular systole. Cardioangiography[23-28] was carried out by direct puncture of the left atrium, demonstrating a narrow communication between the left ventricle and an opacified cavity within the "bulge." The diagnosis of ventricular aneurysm of false type was deemed confirmed.

At operation on Sept. 27, 1955, the mass was seen to be contained within and to be formed by a greatly thickened and adherent pericardium. A purse string suture was placed about a selected site over the fundus of the sac, and the operator's ungloved left index finger was inserted through a stab wound made within its confines. The sac was found to be free of clot and to communicate with the left ventricular lumen by two apertures separated by a few millimeters of ventricular wall from each of which a jet of blood emerged with ventricular systole.

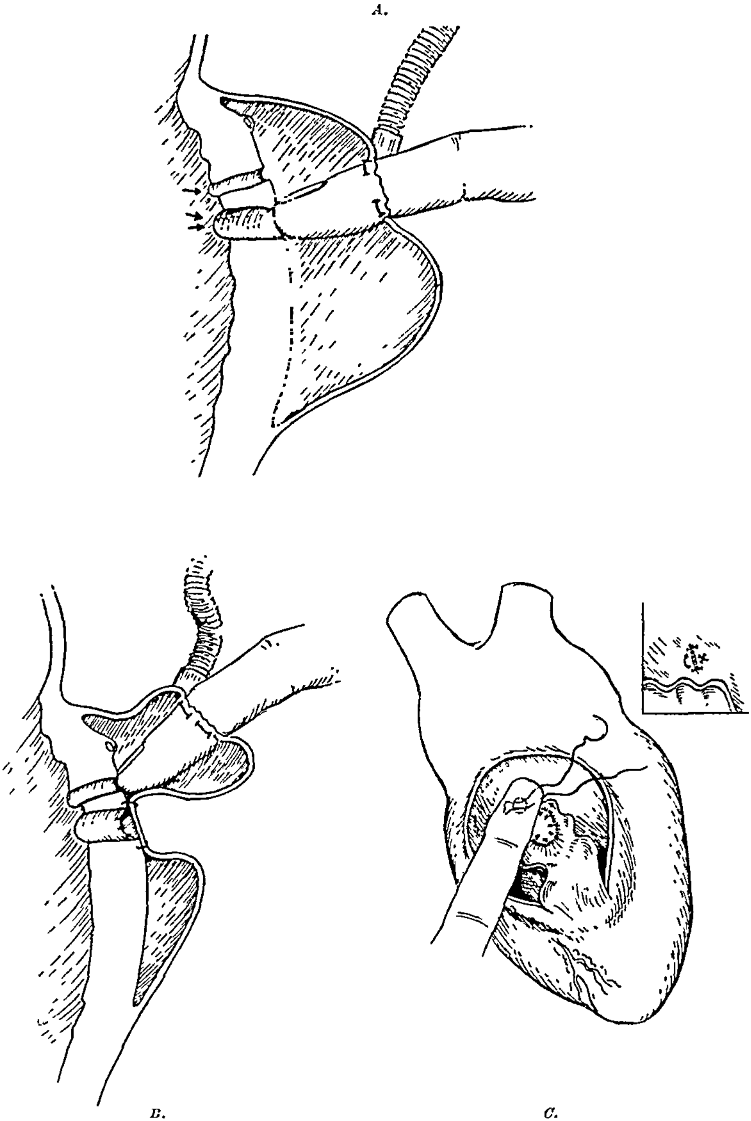


Fig. 5—4, Diagrammatic representation of method digitally examining interior of pseudoaneurysm in G. F There were two openings into the left ventricle from each of which a jet of blood could be felt to emerge with each ventricular systole

B, The flexible aneurysmal wall was sutured down upon the lower opening in order to block it temporarily.

C, Using the tip of the intra-aneurysmal finger, the upper opening was tamponaded while the sac was opened widely. Then this transventricular passage was closed definitively by passing suturing needles under the finger tip, as shown.

(Smith and associates, Surgery 42: 500-501, 1957.)

The exploring finger tip was applied over the lower (larger) opening, blocking its jet. Within a few moments the blood within the sac became coagulated. Temporary sutures of silk were applied between the wall of the sac and the myocardial surface surrounding the lower opening. When they were tied down, the opening became blocked in exactly the same way that an atrial septal defect is closed by suture approximation of its margins to the overlying atrial wall (atrioseptopexy).[29-33]

Now the upper opening was tamponaded with the finger tip and the remaining lumen of the sac was opened boldly. Deep approximating mattress sutures, swedged on curved needles, were applied beneath the finger tip to close this ventricular aperture definitively. The temporary sutures blocking the lower opening were cut away, and this remaining passage was tamponaded digitally. It was sutured then in the same manner as the first (Fig. 5, *A*, *B*, *C*). After repairing the ventricular wall, the mitral valve was explored through an opening made in the appendageal stump and an area of fusion found in the region of the anterior commissure was sectioned with the guillotine knife. Recovery was uneventful and the patient has remained well to date.

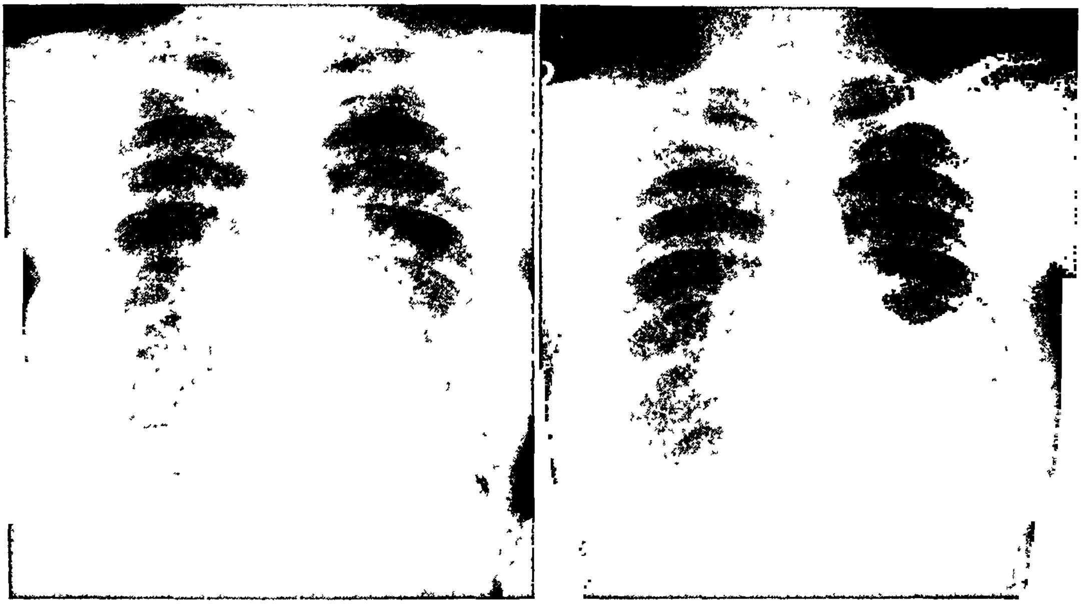

A. *B.*

Fig 6.—*A*, PA roentgenogram taken on E. S. (Case 3) prior to surgery.
B, Marked abnormality of cardiac outline in E. S. observed one year after aortic surgery. (Smith and associates, Surgery, 42: 503, 1957.)

Case 3.—E. S. (Hospital No. 55-14112), a 38-year-old white woman, was operated upon at the Hahnemann Hospital in Philadelphia on Nov. 30, 1953, for combined mitral and aortic stenosis, having the then available transventricular instrumental approach used for the latter valve. Recovery seemed satisfactory and the patient remained in apparent good health until she was involved in an auto accident in July, 1955.

Shortly thereafter she developed atrial fibrillation. X-ray studies at that time showed an abnormal bulge over the left border of the cardiac silhouette. In retrospect, it was decided that a similar but smaller shadow had existed in an early postoperative film (Fig. 6, *A* and *B*).

During the next 3 months, the bulge increased in size and the patient began to suffer from frequent attacks of paroxysmal nocturnal dyspnea. She was re-admitted to the Hahnemann Hospital on Dec. 6, 1955, with a diagnosis of left ventricular aneurysm.

Clinical and physiologic studies revealed a harsh systolic murmur and an early blowing diastolic murmur in the aortic area, and a sharp first sound at the apex (but no mid-late diastolic murmur). The electrocardiogram showed T-wave inversion in Leads I and AVL suggestive of myocardial damage high on the lateral wall of the left ventricle. The left ventricular pressure was 140/10 mm. Hg, the left atrial (mean) pressure was 25 mm. Hg, and the aortic pressure was 126/84. It was felt that mitral stenosis existed, but that no serious obstruction existed at the aortic level.

Cardioangiography by direct left atrial puncture showed an abnormal cavity communicating with the left ventricular chamber.

At operation on Dec. 16, 1955, an ovoid expansile (pulsating) mass the size of a lemon was seen to arise from the left ventricle in the region of the previous operative intervention. It lay beneath and was largely composed of thickened pericardium. The operator's ungloved left index finger was inserted into its lumen through a purse-stringed stab wound as in Case 2. A single communication into the ventricle was recognized. It was tamponaded with the finger tip, the sac was cut away, and the ventricular wall defect was closed with mattress sutures. The mitral valve was re-explored through an incision made in the stump of the left auricular appendage. The valve would admit but a finger and therefore was sectioned at both commissures. Recovery was uneventful.

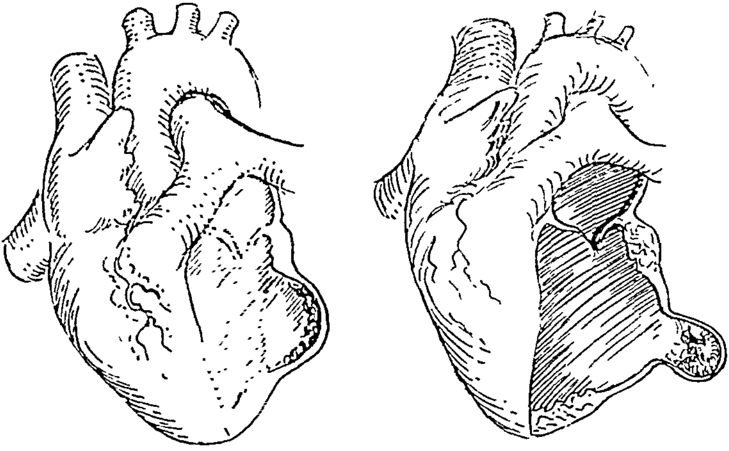

A. *B.*

Fig. 7.—*A*, Diagrammatic representation of diffuse aneurysmal bulge which is the type which characteristically develops in the acute stages of a myocardial infarction. Intraluminal clotting is common.

B, Representation of the thin-walled narrow-necked aneurysm which is characteristic of those which develop late in the course of healing of an infarcted area or as the result of a secondary infarction. Thrombosis is usual within this type of sac.

POSTINFARCTIONAL ANEURYSMS

This group comprises by far the commonest and most important of these lesions. To date, we have operated upon 6 such patients with 5 long-term survivals, including the first historically to recover from excision of a ventricular aneurysm. This case has been reported elsewhere[34] and the operative technique has been described under the very appropriate term of "ventriculoplasty."[34] The abnormality in these cases varies from a relatively di
bulging of a portion of the ventricular wall to a thin-walled, pouchlik
balloonlike structure which communicates with the ventricle b
neck (Fig. 7, *A* and *B*). The former type is the one which develo

stages of a myocardial infarction, and is characterized by the presence of muscular elements in its relatively thick wall. The latter type is a product of the later or chronic stages of a myocardial infarction and is composed chiefly of scar tissue. In either case, the sac may contain clotted material which sometimes becomes calcified. Schlicter, Hellerstein, and Katz[5] found thrombosis in over 50 per cent of their series. This was associated with a 35 per cent incidence of thromboembolic episodes.

The commonest site of localization is the anterolateral aspect of the left ventricle with involvement of the apex in over 50 per cent of the cases. The next most common is the posterodiaphragmatic surface (about 25 per cent). Aneurysmal bulge of the septum is less frequent. The right ventricle is least often involved. Effective treatment of the septal type of lesion would seem to require an open technique with a cardiopulmonary bypass for a proper repair. In this connection, it may be noted that both Cooley[35] and Effler[36] have sutured an acquired defect of the interventricular septum caused by rupture following infarction.

The localization and configurations in our 6 cases were as follows: 2 anterolateral with diffuse bulging including the apex, one localized lateral near the atrioventricular groove, one posterodiaphragmatic, and one huge diffuse lateral mass. There was one double sac with anterolateral and apical bulges.

PATHOPHYSIOLOGY

Due to the semielastic structure of the sac, it becomes stretched out at the height of systole (paradoxical pulsation). The ventricular contraction must raise the intraluminal tension of the sac above the level of the aortic diastolic pressure before ejection can take place. Thus, the dysfunction partakes of that found in mitral regurgitation, a portion of the ventricular content being "wasted" during each beat in distending the abnormal chamber rather than being expelled into the aorta. During the diastolic period, the semielastic sac empties much of its content into the left ventricle; impeding filling from the left atrium. In this respect, the abnormal physiology resembles that of aortic regurgitation.

As a result of this functional inefficiency, the left ventricle is overworked and gradually undergoes compensatory dilatation and hypertrophy. This very enlargement, as Burch and co-workers[37] have shown mathematically, greatly enhances the mechanical handicap. Furthermore, it would seem that the ballooning and outpouching of the aneurysm may exercise a stretching effect upon the adjacent portions of the myocardium during systole, thus contributing to an abnormal and relatively ineffective type of ventricular contraction.

PROGNOSIS

While some aneurysms of the ventricle may be silent and, for a time, pass unnoticed clinically, this is true in less than half the cases. In them, the unexpected onset of arterial embolism may be the first indication of the presence of the lesion. In most cases, the pain of coronary insufficiency persists after the infarction, heart failure develops, and embolic issue or sudden death may punctuate the picture. Schlicter and associates[5] in their study of 102 patients reported a 70 per cent incidence of heart failure, this being the main cause of death in 48.1 per cent. Embolism occurred in 35 per cent and was the main cause of death in 21.7 per cent. Three fourths of their patients died within 3 years, and 88 per cent within 5 years. Actual rupture of the aneurysm is rare,[5, 10, 38, 39, 40] probably because death occurs from some other cause before the dilatation and thinning of the sac becomes extreme. This outlook is dismal in contrast to the average life span of more than 12 years and to the 90 per cent of complete rehabilitation to be expected after uncomplicated myocardial infarction.[39, 40, 41, 45]

DIAGNOSIS

While certain physical signs and certain electrocardiographic findings[5, 29, 41] are considered suggestive of the presence of a ventricular aneurysm, it is our belief that the best and most reliable diagnostic means are radiologic. These include the classic fluoroscopic findings (paradoxical pulsating mass on the border of the cardiac shadow), simple radiographic disclosure of a suggestive bulge upon the cardiac contour, and roentgenkymography. However, it appears that radiographic identification of the opacified left ventricle, preferably by direct left intra-atrial or left intraventricular injection (ventriculography)[23-28] is the surest method of establishing the diagnosis (Figs. 8 and 9). By these techniques, not only the exact anatomic localization of the mass but also the extent and type of involvement and the presence or absence of massive intraluminal thrombosis may be established. The diagnosis of ventricular aneurysm was confirmed in this manner in all but Case 4, in which it seemed evident enough by ordinary radiographic examination.

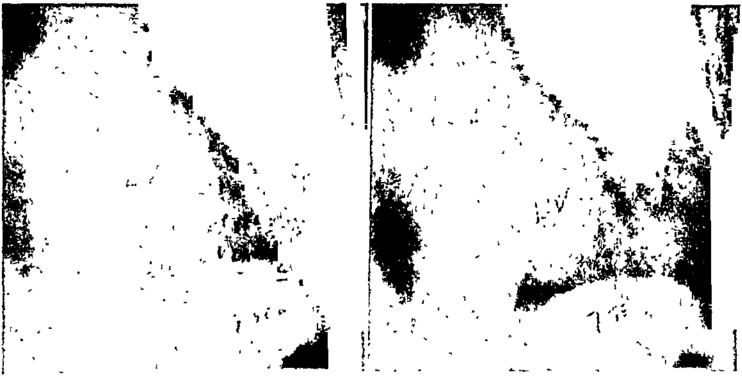
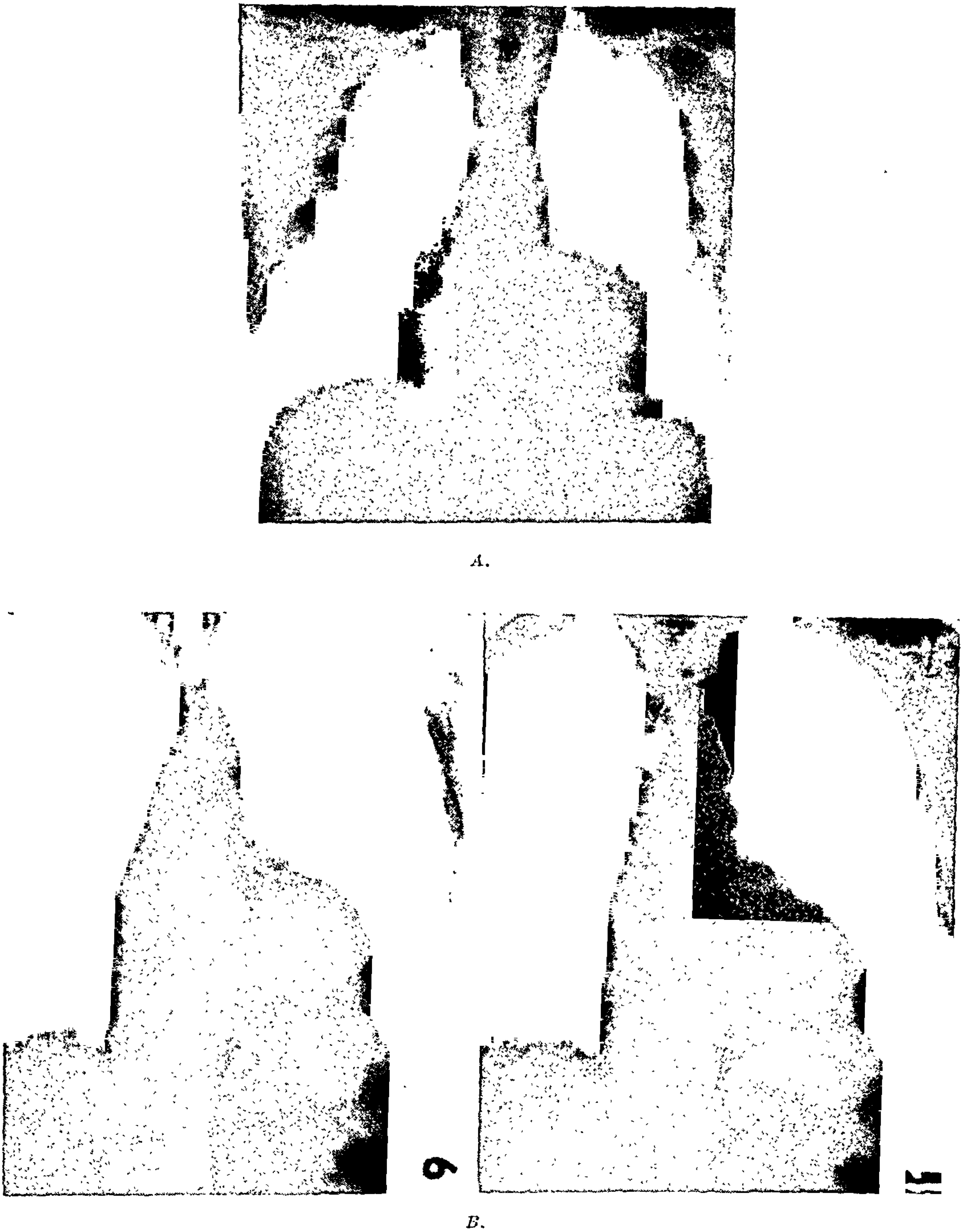


Fig. 8.—By opacification techniques it often is possible to demonstrate the presence and amount of thrombosis which exists within a ventricular aneurysm. (Same case as Fig. 1.)

SURGICAL TREATMENT

In each of our cases, this amounted simply to a subtotal resection of the aneurysm without the use of a cardiopulmonary bypass, enough of the mixed muscular and fibrosed basal tissue of the scar of healing being preserved to



A.

B.

Fig. 9.—Demonstration of a postinfarctional aneurysm later corrected by ventriculoplasty in S. C., a 41-year-old white man. *A*, PA roentgenogram showing nondescript shadow along left ventricular border.

B, Opacification of left ventricle by direct ventricular injection shows an unusual communicating shadow with a flattened top.

permit reconstruction of a ventricular chamber of approximately the normal size and shape for the particular individual. Hence, the term "ventriculoplasty." However, these simple statements compress a great many technical details and the results of considerable clinical, physiologic, and experimental investigation which have not yet been disseminated widely to the profession. That this information may be surgically important is suggested by the fact that we are aware of only 2 long-term survivals among a number of resections for ventricular aneurysm which have been performed by other thoracic surgeons throughout the world. Indeed, some surgeons have chosen to carry out some type of enfolding or plicating operation upon the wall of the sac[43-46] rather than to undertake the presumed great risk of resection.[47-51]

By word of mouth, we have been informed that the causes of death in these clinical cases largely have been of two types. (1) Arterial embolization occurring at the time of operation. Our own single mortality was caused by gross embolization at the moment of compression of the base of the aneurysmal sac in a patient who was known (see Fig. 8) to have had massive clotting within the sac. Since that time, we have worked out a simple and practicable method for the avoidance of this complication. (2) Congestive heart failure during the weeks or months immediately following the operative intervention. While many of these patients may have been in a state of incompletely relieved congestive failure at the time of operation, our experience to date has indicated[50, 51] that immediate improvement is to be expected in this respect following removal of the severe physiologic handicap imposed by the functional existence of the paradoxically expansile mass. True, in some progression or a fortuitous exacerbation of the underlying arterial or myocardial disease might have contributed to a continuation of the downward course. However, it is our belief that in many cases the heart failure must be attributed to a technical error on the surgeon's part. It may be that inadvertently he has included one or more important coronary arterial branches within the suture line, thus aggravating the existing myocardial ischemia. However, it is more probable that his surgical sin has been that of excessive removal of ventricular wall tissue with consequent severe reduction in the size of the left ventricular cavity.[1]

This may seem to be a rather surprising statement since most physicians feel that the cardiac chambers are capable of prompt and extensive compensatory dilatation. While this undoubtedly is true under certain clinical conditions, it appears definitely to be untrue with respect to surgical resection of the ventricular wall. This was brought home to us by the results of an experimental study of the treatment of acute myocardial infarction by excision as suggested by Murray.[52]

EXPERIMENTAL METHOD

Tennant and Wiggers[53] noted that the acutely infarcted area of the heart would beat paradoxically. Murray,[52] in his now classical article, demonstrated that the area could be resected with survival. This acute method of producing a paradoxically beating area in the heart led us to reproduce these experiments in an attempt to solve the problems attendant upon the resection of ventricular aneurysms. They also led to an interesting observation on the animals when large areas of acutely infarcted muscle were removed, which was in contrast to the previous work.

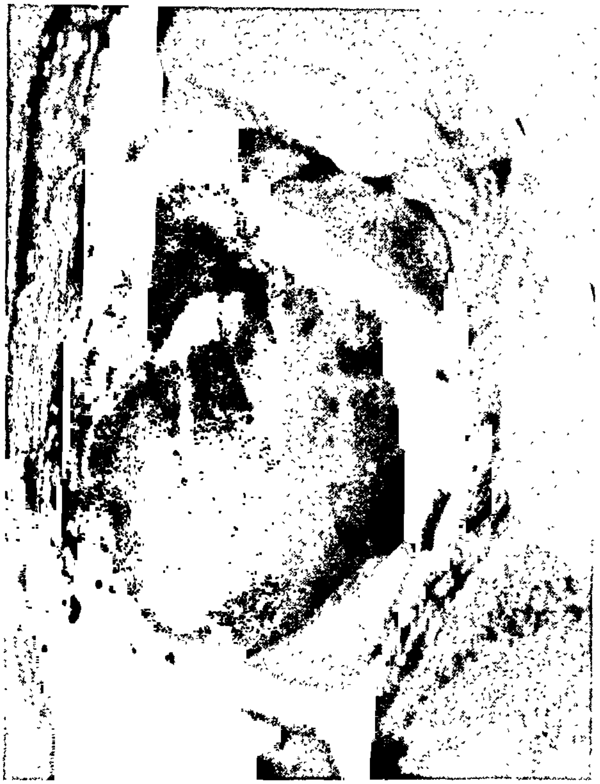


Fig. 10.—Photograph showing the cyanotic infarcted paradoxically pulsating area of left ventricular wall which develops following ligation of the anterior descending branch of the left coronary artery in the dog.

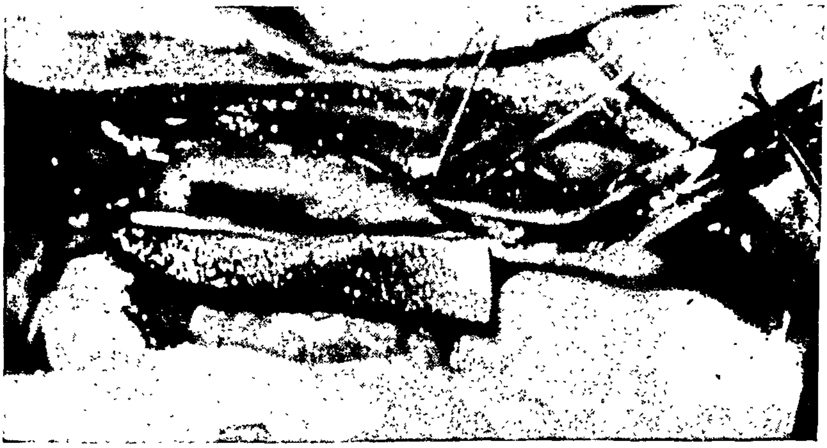


Fig. 11.—Application of the Rumel-Morse clamps armed with strips of Ivalon sponge to exclude the infarcted portion of the left ventricle.

Initial attempts were made simply to place mattress sutures under the infarcted area, tie them down, and then excise the infarcted area. It was noted that when a large area such as that produced by ligation of the anterior descending branch of the left coronary artery was involved, the tension necessary to coapt the edges would cause them to cut through the muscle with consequent severe hemorrhage. Further, there was no provision in this technique for the evacuation of clots such as might occur in aneurysms. Therefore, a method for evacuation of clots and for buffering the suture line was devised.

Twenty-five mongrel dogs, weighing from 5 to 15 Kg., were prepared. The anterior descending coronary artery was ligated 2 to 3 cm. from the point where it branched from the main left coronary. This site was chosen to avoid possible inclusion of the septal artery. The subsequent infarction involved at least one half of the anterior surface of the left ventricle and the apex (Fig. 10). A Rumel ventricular clamp, as modified by Morse,[34] was armed with Ivalon strips placed so that the teeth protruded through the sponge (Fig. 11).

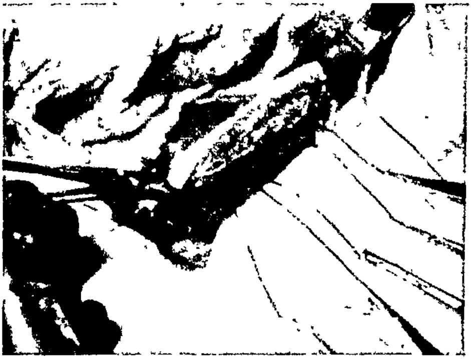


Fig. 12.—The approximating sutures are passed through the Ivalon strips after amputating the infarcted muscle.

These were applied at the limits of the infarcted area into the normal muscle so that the teeth were engaged into but did *not* penetrate the wall. A large incision was made into the infarcted area and the wound was allowed momentarily to bleed. By this method any contained clots would have been "flushed-out." The bleeding could be controlled instantaneously and completely by closing the clamp. The Ivalon sponge prevented its jaws from cutting through the wall or injuring it. The clamp was undersewn, using interrupted, overlapping mattress sutures placed directly through the *Ivalon*. The excluded portion of the ventricle then was cut off above the sutures. The clamp was removed and the Ivalon sponge left permanently in place (Fig. 12). If the mattress sutures are placed properly so that they overlap, no additional sutures are needed. This method appears to overcome most of the technical problems concerned with the resection of a ventricular aneurysm.

As the idea of excision of areas of myocardial infarction has been postulated and the opinion exists that the time for resection may not be after the aneurysm has formed but immediately after the acute infarction, the dogs were saved for further study.

All of the dogs had one half or more of the anterior surface of the left ventricle infarcted. This was represented by a triangular area which on excision measured 3 by 3 by 3 cm. or more. A rim of 1.5 to 1.0 cm. of tissue surrounding this area was incorporated within the suture line and thus was excluded from the functioning myocardial musculature. Six of the dogs died

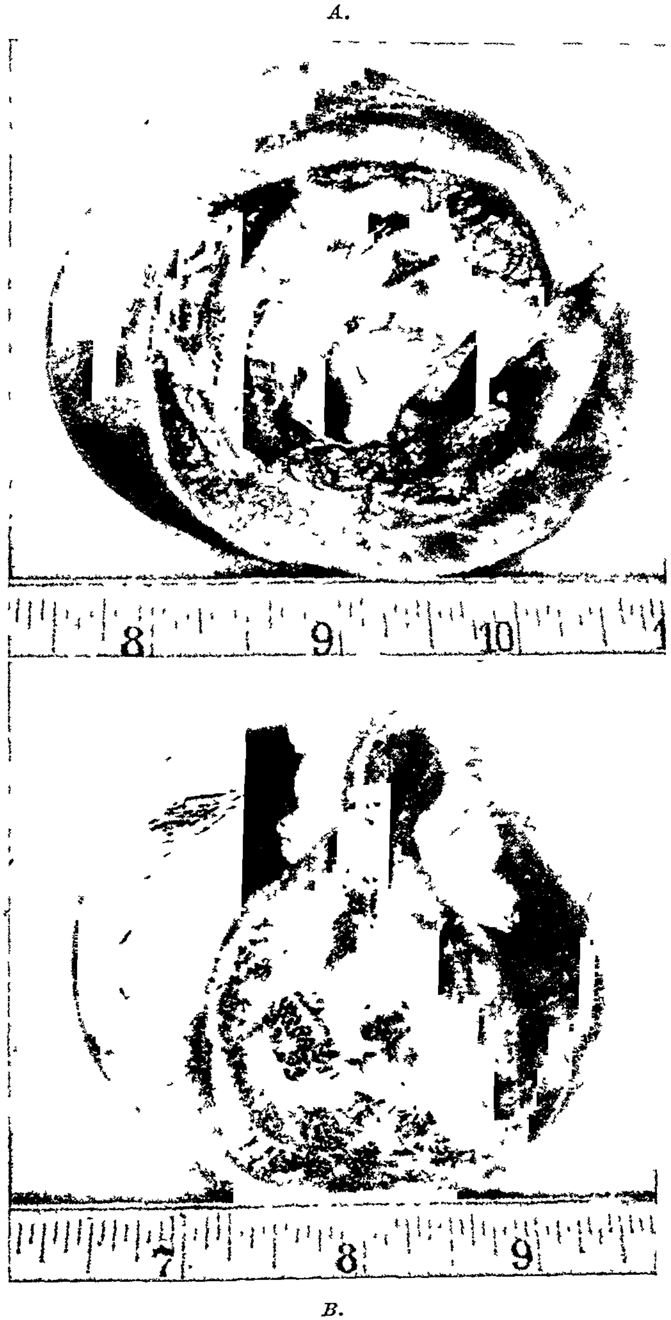


Fig. 13.—*A*, Cross-sectional view of the normal dog heart taken one third of the distance from the apex of the ventricle toward the base.

B, Similar section of heart of dog subjected to excision of an experimentally created infarct 13 days previously. Note the marked reduction in the size of the left ventricular chamber.

immediately from irreversible ventricular fibrillation. The hearts of two additional dogs fibrillated, but a normal sinus rhythm was re-established by electric countershock. Two dogs died of bleeding when the sutures were not overlapped properly. One died of empyema. Thirteen dogs died from the first to the ninth day with congestive heart failure. They exhibited pulmonary edema, engorgement of the liver, hydrothorax, and ascites. Three dogs were sacrificed on the tenth, thirteenth, and twenty-second days. They had similar findings. The longest lived dog in the series had massive ascites and hydrothorax and was deteriorating when sacrificed.

It had been noticed that immediately after the clamp was closed, there was a moderate to marked dilatation of the left auricle, the right ventricle, and the pulmonary artery. There was no immediate drop in blood pressure after the heart was infarcted[55, 58] but a gradual drop of 10 to 30 mm. Hg was observed after the clamp was applied.

The hearts were studied at post mortem. The inside of the heart showed only a narrow linear scar representing the site of excision. There was no evidence of mural thrombosis. On the outside, there were dense pericardial adhesions to the resected area, giving it a smooth surface. The suture line was represented by a tough fibrous scar. In the dog that was sacrificed at 22 days, the Ivalon sponge was well infiltrated with fibrous tissue. In no instance was a papillary muscle destroyed. When it was infringed upon it was caught up within the suture line. At no time was there evidence of mitral insufficiency as a result.

These hearts did not appear to show any compensatory dilatation to compensate for the excision of ventricular tissue. There was a reduction in the ventricular volume which appeared to be about half of its usual capacity (Fig. 13, *A* and *B*). These findings were consistent in all the surviving dogs.

DISCUSSION

The net conclusion from this work is that the heart is not capable of free and unrestrained dilatation and that this cannot be expected promptly after surgical reduction in the size of the left ventricle, significantly below normal limits. Instead there is marked restriction in the stroke and total cardiac output and a type of circulatory obstruction similar to that observed in experimentally produced mitral stenosis.

While the considerable increase in left ventricular volume which is characteristic of hearts with myocardial aneurysm would seem to militate against easy production of such functional inlet obstruction, it is evident that there must be a limit even in these cases to the amount of permissible ventricular wall resection. After all, the predominant source of such aneurysms is the very large infarction which is caused by occlusion of a major coronary arterial branch comparable to that ligated in the experimental animals. Since the normal process of healing of an area of myocardial infarction is by fibrous replacement, it is clear from the usual benign course in uncomplicated clinical cases that the presence of such areas of scarring, per se, are not deleterious to heart function. Their presence does not necessarily imply an enlargement of the ventricular lumen since they merely may replace an equal area of destroyed muscle tissue.

Since the aneurysmal sac or pouch characteristically represents but the thinned-out central portion or fundus of the postinfarctional scar, it would seem logical merely to amputate this bulging area and to use the remaining peripheral portion of the scar for closure of the created ventricular wall defect. The combined fibromuscular structure of this peripheral portion of the original area of healing represents a nearly ideal tissue for suturing, being both strong and supplied with a rich capillary type of vasculature (augmentation of the intercoronary artery communications).

We, therefore, conceive it to be the surgeon's responsibility in a case of ventricular aneurysm to remove such portion of the postinfarctional scar as is necessary to overcome the deleterious physiologic effects of the aneurysm and, at the same time, to preserve sufficient of its periphery to reconstruct at least a minimally adequate ventricular chamber. In other words, he must "tailor" the ventricular cavity to as near the normal size and shape as is feasible. Such concepts are implicit in the term ''ventriculoplasty.''

We believe that excessive removal of the scarred portion of the ventricle may have been responsible for the development of persistent heart failure in some of the reported late deaths following excision of ventricular aneurysms.

It, also, is manifestly the surgeon's responsibility to avoid inadvertent dislodgment of clotted material which may be contained within the aneurysmal *pouch during the manipulations incident to its removal. While this might* be accomplished by the use of an open technique and a heart-lung bypass, we feel that it may be done more simply and with equal effectiveness by the use of the ''flush-out'' technique as described.

ILLUSTRATIVE CASE REPORTS

CASE 4.—V. F. (Hospital No. 54-04387), a 56-year-old white male Brazilian coffee planter, was admitted to the Hahnemann Hospital in Philadelphia on April 8, 1954, with a history of a myocardial infarction in January, 1953. In May of 1953, he had ventricular failure and at this time a diagnosis of left ventricular aneurysm was made. This was controlled by medical therapy and the patient did moderately well until December, 1953, when he again went into failure which persisted in mild form in spite of vigorous medical therapy.

On admission to the hospital, the patient was dyspneic at rest and orthopneic. The blood pressure was 124/80 mm. Hg. A diastolic gallop rhythm was present. The heart sounds were distant but no murmurs were heard. There were moist râles at both bases, the liver was palpable three fingers below the costal margin, and there was a one plus ankle edema. The electrocardiogram showed evidences of an old anterior wall infarction. The roentgenogram demonstrated a bulging on the anterolateral surface of the left ventricle. A diagnosis of an old anterolateral infarction with a ventricular aneurysm was reached and the patient was prepared for surgery. In spite of digitalis, bed rest, mercurial diuresis, and a salt-poor diet the gallop rhythm and dyspnea persisted.

On April 15, 1954, a left posterolateral thoracotomy was performed. An anterolateral infarction was found, and a ventriculoplasty was performed (Fig. 14). The patient tolerated the procedure well and left the operating room in good condition. The diastolic gallop disappeared on the first postoperative day, was heard intermittently during the first week, and then disappeared.

The patient was kept at bed rest for 5 weeks and then observed for 7 weeks. During the latter period he was able to walk and to climb three flights of stairs without dyspnea.

The patient was re-admitted to the hospital for check up in December, 1956. His only complaint at this time was of occasional bouts of deep and fast breathing which occurred during sleep, causing him to awaken.

He had no evidence of failure or of further cardiac deterioration. However, he was still maintained on digitalis.

Comment: This man, who was, historically, the first patient to survive ventricular resection for cardiac aneurysm,[13, 34, 56] was practically terminal at the time of surgery. Yet his operative response was gratifying and he remains well to date. At check up 2½ years after resection of the ventricular aneurysm, he seemed to be following the usual relatively satisfactory course to be expected of a patient who had suffered a myocardial infarction without complication. He continues to do well 3 years following operation (Fig. 15, *A-D*).

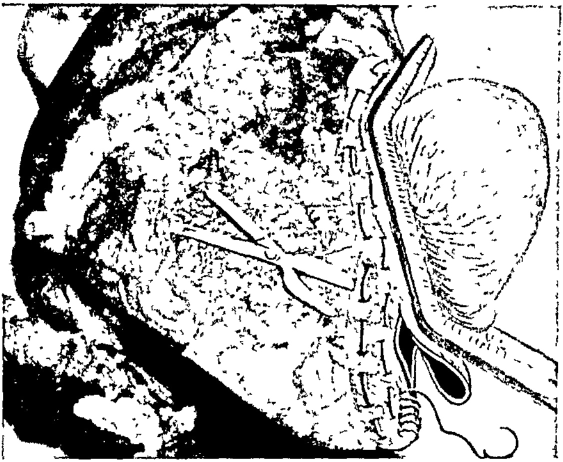


Fig. 14.—Semidiagrammatic representation of technique of ventricular excision employed in V. F. (Bailey, Surgery of the Heart, ed. 1, Philadelphia, 1955, Lea & Febiger, p. 1045.)

Case 5.—R. T. (Hospital No. 56-19642), was a 56 year old white woman who, on April 30, 1955, had a sudden onset of severe chest pain. This was diagnosed as a myocardial infarction and the patient was hospitalized for 2 months. During July, 1955, enlargement of the cardiac silhouette was noted on x-ray studies and the patient was placed on digitalis therapy. In December, 1955, a bout of atrial fibrillation occurred, accompanied by ankle edema and shortness of breath. This was treated with quinidine. She improved somewhat during the next few months but still had marked limitation of activity because of shortness of breath. The patient was admitted to the Hahnemann Hospital for evaluation.

There was no neck vein distention, the lungs were clear to auscultation and percussion. The heart exhibited a normal sinus rhythm, the sounds were diminished in intensity, and there were no murmurs. The blood pressure was 104/60 mm. Hg. The liver was not palpable and there was no ankle edema.

The electrocardiogram showed findings indicative of a previous anterolateral myocardial infarction. Routine x-ray studies showed a bulging and elevation of the superior portion of the left ventricle, with a discontinuity of its border. Cardioangiography, by left atrial puncture, demonstrated a large ventricular aneurysm (Fig. 16, *A* and *B*).

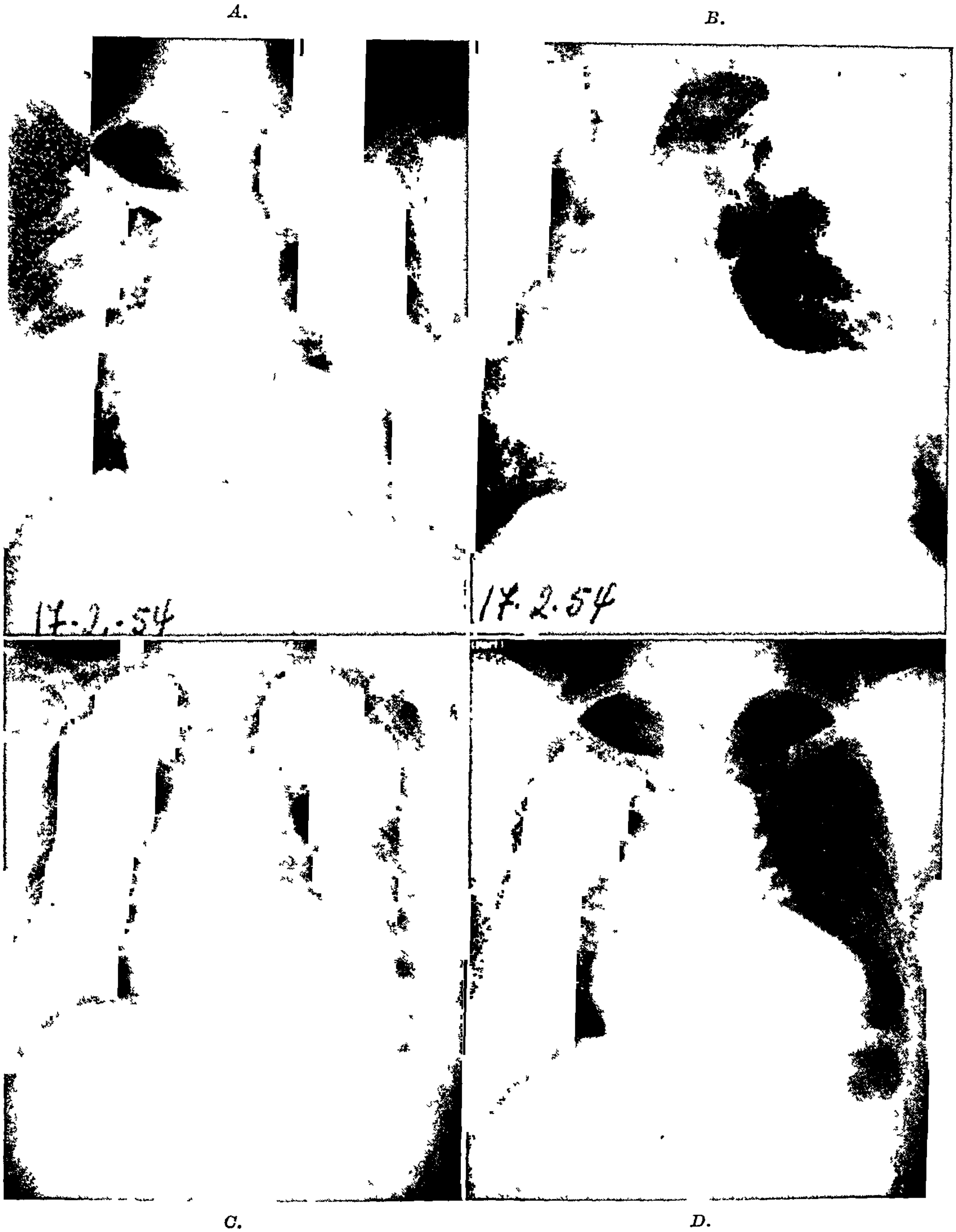
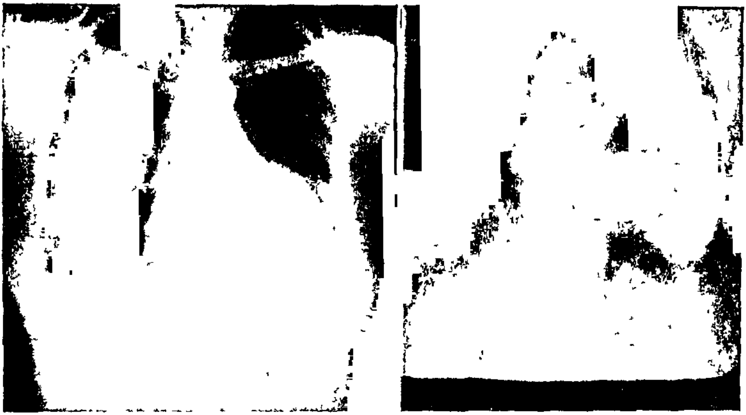


Fig 15.—*A* and *B*, Preoperative PA and oblique roentgenograms of V F. (Case 4) showing a bulging of the left border of the heart which was diagnosed as a postinfarctional ventricular aneurysm (Bailey, Surgery of the Heart, ed. 1, Philadelphia, 1955, Lea & Febiger, p. 946.)

C, PA roentgenogram of the heart of V F., 6 weeks after ventriculoplasty.

D, Comparable roentgenographic study of V F. taken 2½ years postoperatively.

The patient was prepared medically and operated upon on April 30, 1956. After the induction of anesthesia it was noted that the carotid pulse was absent. The left chest was opened quickly and the heart was found to be in standstill. Cardiac massage was instituted and prompt restitution of the heartbeat was achieved with restoration of the blood pressure to a systolic level of 110. After recovery seemed assured, resection of the aneurysm was undertaken and a ventriculoplasty was performed. The patient tolerated the procedure well and left the operating room in good condition. The patient had an unremarkable convalescence and left the hospital on the seventeenth postoperative day. Her condition today is reported as significantly improved.



A. *B.*

Fig. 16.—*A*, PA roentgenogram of female R. T (Case 5) showing abnormal cardiac silhouette.

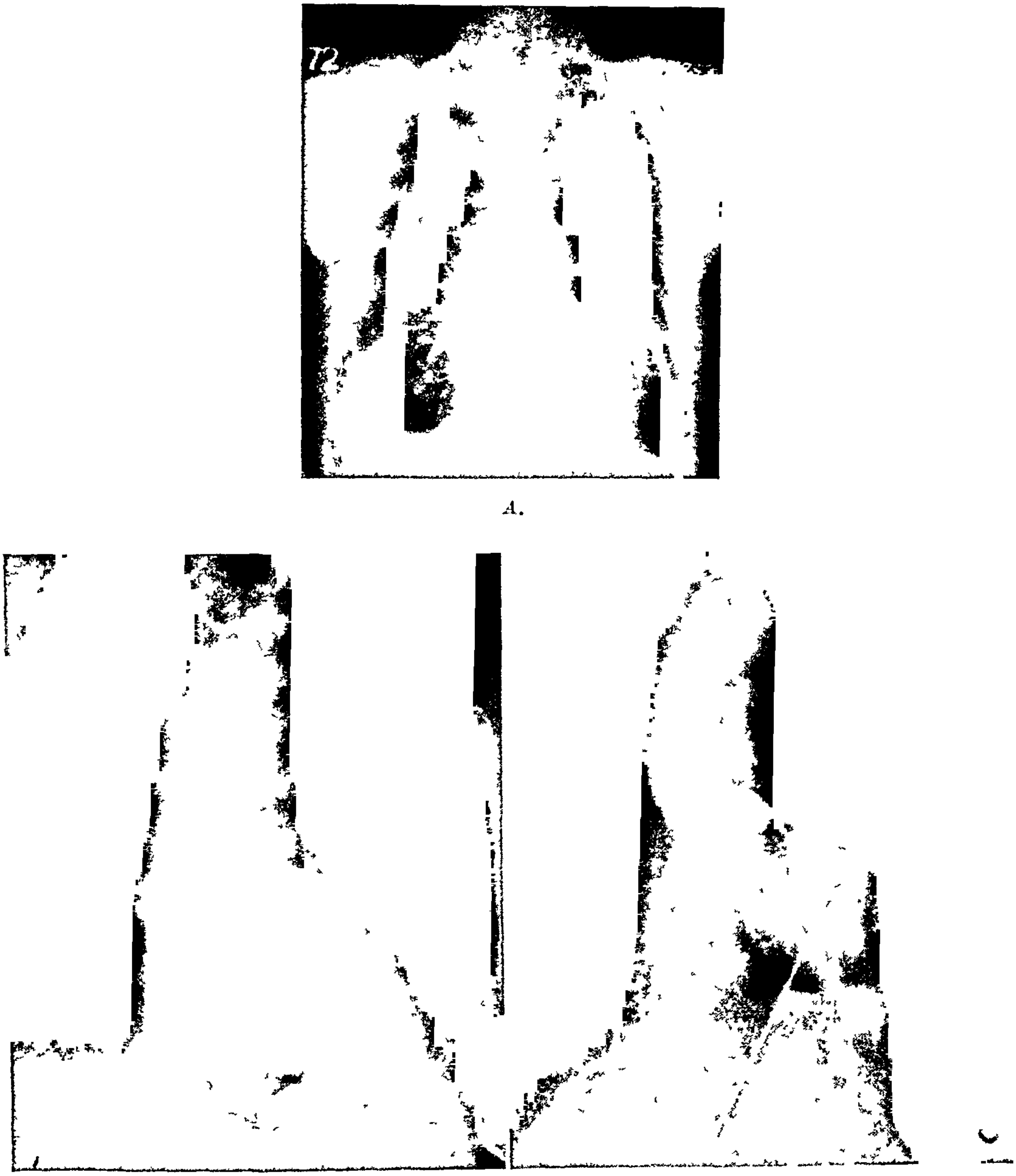
B. Cardioangiogram obtained by left atrial injection showing large anterolateral aneurysmal shadow superimposed upon the shadow of the interior of the left ventricle (outline of latter indicated by dotted lines).

Case 6.—R. T. (Hospital No. 56 28430), a 65-year old white man, was admitted to Hahnemann Hospital on Dec. 2, 1956. In March, 1955, the patient had had an acute myocardial infarction for which he was hospitalized. Prior to this episode he had had some substernal discomfort not definitely associated with exertion. Following the infarction, he developed easy fatigability and dyspnea on exertion.

On admission the blood pressure was 120/70 mm. Hg. The lungs were clear to auscultation and percussion. The heart sounds were distant. No murmurs were discernible. The liver was not palpable and no ankle edema was present. The electrocardiogram showed changes indicative of extreme myocardial damage involving the anteroseptal portion of the left ventricle. Routine roentgenograms showed a bulging of the left ventricular wall which was believed to represent an aneurysm (Fig. 17, *A*). Ventriculography clearly demonstrated a large left ventricular aneurysm with a double pouch (Fig. 17, *B*). The patient was prepared for surgery with digitalis, Thiomerin, and bed rest.

At thoracotomy, on Dec. 17, 1956, two bulges were found adjacent to each other. The first was on the lateral aspect of the heart. The second was adjacent to the first in the region of the apex. Using the technique of ventriculoplasty, both aneurysmal bulges were resected. The patient tolerated the procedure well.

Postoperatively, the patient did well until the third day when he had a brief hypotensive episode which required a continuous infusion of norepinephrine. Subsequently, 650 c.c. of sanguineous fluid was aspirated from the left chest. No further difficulties red, postoperative ventriculogram revealed a nearly normal shaped left ventricle (Fig. 17, *C*). The patient was discharged 4 weeks postoperatively in good condition. A recent medical report is most satisfying, both dyspnea and fatigability being relieved.



A.

B. *C*

Fig. 17.—*A*, PA roentgenogram of male R T. (Case 6) showing aneurysmal bulge upon the anterolateral aspect of the left ventricle

B, Preoperative ventriculogram (by direct ventricular injection) obtained from Case 6 showing double aneurysmal bulge.

C, Postoperative ventriculogram in Case 6 showing effect of ventriculoplasty upon left ventricular chamber

TECHNIQUE OF VENTRICULOPLASTY

Depending upon the site of localization of the aneurysmal dilatation as determined by fluoroscopy and cardioangiographic opacification, the operative approach is made through a left anterolateral intercostal (fourth or fifth) incision for anteriorly or anterolaterally located aneurysms, or through a left posterolateral one for a lateral, posterolateral, or diaphragmatic projection, in this case using the fifth, sixth, or seventh intercostal space (whichever seems most appropriate). After the pericardium is exposed, it may be incised and dissected away from the aneurysmal bulge, the integrity of the left phrenic nerve being preserved.

When the aneurysm has been exposed as completely as possible, the manner in which the ventricle is to be tailored is determined. In most cases, this will amount to simple longitudinal application of the Rumel-Morse clamp to the basal portion of the aneurysm in such a way that after the fundus of the aneurysmal bulge has been amputated the contour of the repaired heart wall will be restored as nearly as possible to normal. Careful inspection and palpation of the surface of the heart will indicate the possibility or likelihood of dividing or occluding by the suture line any sizable functioning coronary arterial branches, and also will disclose any area of unusual thinning of the ventricular wall which would be retained should the proposed line of resection fail to include it.

The operator's ungloved right index finger is inserted into the left atrium by way of an incision made into the purse-stringed tip of the left auricular appendage. At transient strategic intervals the finger tip is advanced through the (normal) mitral valve to explore the interior of the left ventricular cavity and perhaps that of the aneurysm itself. The localization of the sites of insertion of the papillary muscles with respect to the aneurysm is investigated. If excessive or loosely attached thrombotic material is present, suggesting the danger of partial dislodgment at the moment of clamping of the base of the aneurysm, especial care is taken with the steps of the "flush out" technique.

Having determined the extent and location of the portion of the sac to be resected, the special Rumel-Morse clamp is applied with a safe margin so that the long teeth upon its opened jaws penetrate deeply into the partly fibrosed ventricular wall. After everything is in readiness, blood transfusion having been administered to a slight excess, the portion of the aneurysm to be resected is suddenly and widely incised, permitting a momentary massive gush of blood from the ventricular cavity which tends to flush out any loosely attached clot within the sac. Then, the jaws of the clamp are approximated, abolishing further blood loss and restoring the functional integrity of the circulation (Fig. 18, *A-D*). If necessary, further transfusion of whole blood is administered.

The selected portion of the now excluded fibrosed ventricular wall is excised. Any visible, partially adherent, thrombotic material may be extracted from between the protruding ventricular lips. These are approximated with a row of interrupted interlocking mattress sutures of heavy silk placed either above or below the clamp at a distance of 8 to 10 mm. from the cut margin. The cut edges are oversewn with a continuous suture of medium silk. In cases or in areas where there has not been sufficient fibrous infiltration of the residual wall to render it tough and strong, the suturing needles may be passed through strips of compressed plastic sponge,* 2 cm. wide and 3 mm. thick. This material acts as an excellent buffer between the epicardium and the suture loops which otherwise might tend to cut through.

After the line of repair has been completed, the clamp is released a little at a time until the two rows of sutures are restraining the full intraventricular pressure. If any bleeding occurs from the suture line, one or more additional mattress stitches may be placed to control it, preferably without re-application of the clamp.

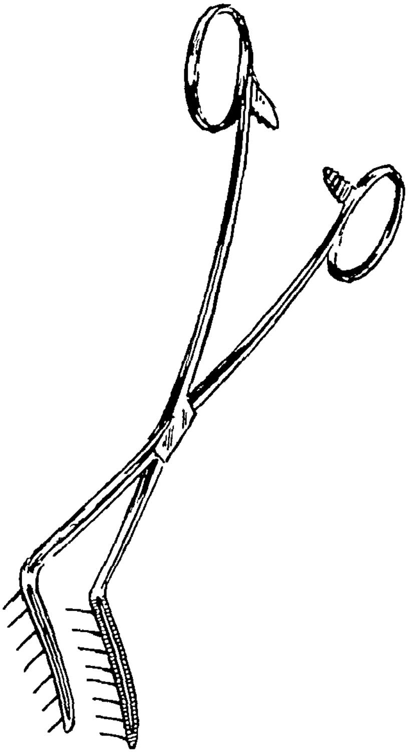


Fig. 18.—*A*, Rumel-Morse fenestrated and toothed ventricular clamp. (Surg., Gynec. & Obst. 104: 539-542, 1957.)

If enough pericardial tissue remains to cover the site of ventricular repair, it may be so utilized. However, an adequate and dependent pericardial opening must be provided to permit free drainage of serosanguineous exudate into the left pleural space. The latter is drained into a water-seal bottle by a multiwindowed No. 28 Fr. urethral catheter inserted through a stab wound made in the seventh or eight intercostal space in the posterior axillary line.

The thoracic wall is repaired in layers, using pericostal and fascial sutures of No. 2 chromicized catgut. The skin is closed with continuous running or subcuticular stainless steel wire.

The method of repair of a true ventricular aneurysm by an open technique, as suggested by Lillihei,[57] does not differ greatly from this except for the necessity for cannulation and the operation of the cardiopulmonary apparatus. Complete circulatory bypass is preferable to merely a left-sided one. With this technique, the special Rumel-Morse clamp is not necessary, the

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*Ivalon: Obtainable from Davis and Geck, Danbury, Conn.

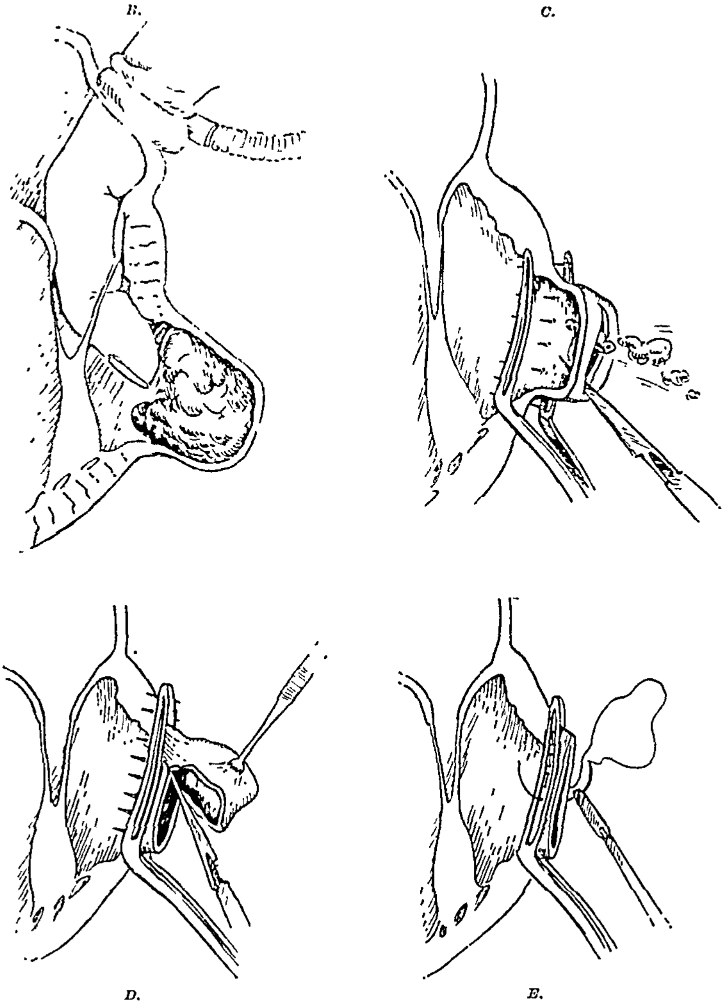

Fig. 18 Cont'd.—*B*, Operator's ungloved right index finger is inserted into the left ventricle by way of the left auricular appendage and the mitral valve to explore the chamber and to detect the presence of any loose or excessive thrombotic material.

C, The teeth of the clamp are applied securely along the selected line and the fundus of the unclamped aneurysm is incised widely to "flush out" any loosely attached thrombotic material.

D, The jaws of the clamp are closed to restrain bleeding and the excluded aneurysmal wall is trimmed away.

E, The ventricular lips are approximated by mattress sutures applied through the slots in the fenestrated jaws of the clamp. After removal of the clamp, the cut edges are oversewn. (Bailey and Gilman, Surg., Gynec. and Obst 101: 539-542, 1957. Courtesy of the Franklin H. Martin Memorial Foundation.)

selected portion of the bulging ventricle simply being excised and the interior of the chamber being exposed. Any clots which extend into the left ventricle are removed by suction or extracted mechanically.

After displacing the air from the open left ventricular (and atrial) chamber by gaseous carbon dioxide (in order to obviate the possibility of air embolization), the cavity is filled with blood or saline, and the heart wall is repaired in a manner similar to that described for the closed method.

SUMMARY AND CONCLUSIONS

With the increased incidence of surgical ventriculotomy, we may expect in the future to encounter more ventricular aneurysms of traumatic (false) type. While every effort should be made to prevent their development, we feel that they may be corrected with relative safety by the technique herein described.

Since adopting the "flush-out" technique, following the death of our second postinfarctional patient from embolization, and by continuing the use of the principle of ventriculoplasty (tailoring the ventricle) rather than attempting totally to excise all scarred tissue, there has been a gratifying surgical and clinical response from each patient so operated. In view of the fact that no "revascularization procedure" was included in the operative technique, it is believed that all observed benefit was due to the removal of the physiologic burden imposed by the paradoxically pulsating sac itself. We *feel* that a widespread application of these concepts and techniques will enable others to achieve equally good results.

In view of the gloomy outlook for individuals with symptom-producing postinfarctional ventricular aneurysm, and the rather dramatic response to appropriate operative intervention, we strongly urge surgical consideration for these common, although less often recognized, sequelae of coronary arterial disease. Knowledge that an effective surgical method of treatment exists no doubt will stimulate an increased awareness on the part of the internist that such a lesion may exist. By appropriate opacification techniques the diagnosis may be established with certainty and the size, shape, and localization of the aneurysm determined.

It is to be hoped, in the future, that these patients may be referred for surgery at a somewhat earlier stage in their devolutionary course when the chances for survival and clinical "cure" will be greater, rather than in the near-terminal or pre-terminal state in which we have seen them to date.

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DISCUSSION

DR. EGBERT H. FELL, Chicago, Ill.—At the Cook County Hospital, Dr. B. M. Gasul and his associations had an opportunity of studying a 7-month-old, healthy looking infant who had a loud machinery murmur at the level of the cardiac apex.

(slide) The angiocardiogram of the right side of the heart is normal, but on the left side of the heart we see a very large right coronary artery filled with dye ending in an aneurysmal-like swelling at the apex of the heart.

(movie) We have had one other patient with an anomalous coronary artery entering the right ventricle just proximal to the pulmonary valve with no aneurysmal dilatation, and a preoperative diagnosis of patent ductus arteriosus. Here we have a true arteriovenous aneurysm at the apex of the heart; the movie shows the aneurysm very well, and you see the large right coronary artery coming up from behind. The coronary artery was clamped, divided, and sutured. Blood now enters the aneurysmal sac from the right ventricle. The coronary artery was every bit of four times its normal size. The wall of the aneurysm was resected and the ostia are seen, one from the coronary and one from the right ventricle. The ventricular defect was closed in two layers. Pathologic studies of the resected material confirmed it to be ventricular wall. The child is now about 2 months postoperative and is doing well.

This movie has shown an unusual experience we have had at the Cook County Hospital with a congenital arteriovenous fistula of the right coronary artery and right ventricle with an aneurysm of the apex of the right ventricle. It was diagnosed prior to surgery and treatment resulted happily in a cure.

DR. J. L. MANGIARDI, Garden City, N. Y.—I wish to congratulate Dr. Bailey for his excellent results in pioneering a new operation and to add a case report.

(slide) This is one of the 25,000 cases available in 1956 that Dr Bailey mentioned This 41 year old man presented with paradoxical pulsations over the aneurysm and severe hypertension ranging from 170 to 250 systolic He had no history of coronary artery occlusion, but the electrocardiogram was compatible with an anterior wall infarction and aneurysm

(slide) This is a slide taken 2 years prior to admission and shows no evidence of aneurysm at that time so that, with the electrocardiogram, we felt quite confident of our diagnosis of postinfarction myocardial aneurysm

(slide) We had just read Dr Bailey's original report of his first successful case, and decided to operate on this patient This shows technique of resection—but it is not absolutely correct, we actually had three suture lines, the first a continuous mattress suture, the second an interrupted layer, and the third sutured over and over with fine silk exactly as shown in the previous slide Before resection, I might mention that we thought the aneurysm was full of clot and we had in mind to stab the apex, allow clot to extrude, and then clamp the base However, we were very fearful that our reflexes would not measure up to Dr Bailey's criteria, so we did not do it The base was simply cross clamped, and although an embolus was thrown off, it was fortunately very small, as the patient recovered rapidly and is now well in all respects, a year and a half postoperatively

(slide) Here is the specimen This is a centimeter rule Looking at the aneurysm from within its cavity, you can see a large amount of clot and fibrin, there was more on the specimen at the time this was taken off

I was a little surprised at what Dr Bailey had to say about muscle in the wall of the aneurysm We were surprised and very disappointed because we did not find muscle in the wall of this resected area

(slide) This shows the microscopic appearance of the aneurysm It has been of great interest to us that this man's hypertension has never risen above 160 ever since the time of resection of this aneurysm So far as the hypertension is concerned, the only thing I can think of is the decrease in size of the left ventricular cavity, but of course this remains a question

(slide) We have had a lot of cardiographs in the follow up of this case, and our cardiologist is much interested in their comparison This is the immediate preoperative study, and I think you can see the high S T segments in Leads I and AVL particularly, and inverted T waves

(slide) This is an immediate postoperative cardiogram The S T segments are now normal, and the T waves are flat Very little change has occurred since

DR LEWIS H BOSHER, JR, Richmond, Va—We have encountered still another type of myocardial aneurysm which because of its rarity is perhaps worth recording here This was the case of a 33 year old Negro woman who entered the hospital in chronic cardiac tamponade She had an elevated venous pressure of more than 250 mm of saline, a depressed blood pressure and pulse pressure, and a very large liver The cardiac silhouette was quite large, aspirations of the pericardium were done which yielded almost frank blood This temporarily relieved the cardiac tamponade Because of the rather marked abnormality of the clotting mechanism, operation was temporarily deferred She had a very low fibrinogen, and this was corrected by intravenous fibrinogen At operation several days later, this was encountered

(slide) The pericardial sac was markedly enlarged and had a large quantity of bloody fluid present in it On the wall of the right atrium very close to the atrioventricular groove there was a saccular, paper thin aneurysmal dilatation about 4 cm in diameter The atrium was explored as we thought that perhaps some type of intracardiac tumor was present, but none was found The wall of the right atrium itself seemed somewhat dilated, perhaps due to tricuspid insufficiency This was not definitely established The wall was quite friable next to this sacculation

(slide) At the top of the slide you see the paper thin aneurysm which bled on contact with the fingers and which sealed spontaneously (slide) A slab of polyvinyl sponge was sutured down over the aneurysm and this concluded the operative procedure. No cardia was found in the pericardial fluid and thus this represents a mycotic aneurysm. This patient's low-grade fever did not subside postoperatively until after an intensive course of therapy with sulfadiazine. She remains well 6 months later.

DR. JOHN C. JONES, Los Angeles, Calif.—I enjoyed Dr. Bailey's discussion and I merely arise to tell you that I came unprepared to discuss my one case. But I bring up this one point. This case of mine was operated upon before Dr. Bailey published his technique, and I do not believe that whether or not you anticipate there is a clot you should put a finger inside to release the clot before you get a chance to resect the aneurysm. The other point is that I do not believe the best technique is to put a clamp on these ventricles. In the first place you will probably ruin your clamp, and in the second place it is not good technique anyway.

I think as we confront this problem, the best way to resect an aneurysm is to place the sutures first, resect the aneurysm, and then draw up the sutures and tie them, and then you will have no difficulty; you have a clot that will come out, if you do not have a clot you are not in trouble, and if you put your finger inside you are likely to release the clot.

The patient I operated upon was in severe decompensation, he had been decompensated for years, and he is now particularly well compensated and has done very well. Just the simple manipulation of placing the sutures, resecting the aneurysm, and tying them, was effective.

DR. DENTON A. COOLEY, Houston, Texas.—I think from the standpoint of completeness it may be worth while to state that there are other complications of coronary occlusion and myocardial infarction which at this time may be surgically correctible. I refer to the acutely perforated ventricular septum and to the ruptured papillary muscle. Both of these are highly fatal complications of coronary disease but, with the methods of cardiopulmonary bypass at our disposal, they could be handled successfully in some instances. We have had experience with one patient with perforated ventricular septum who went through the operation satisfactorily. Unfortunately he died 6 weeks later of pericarditis and empyema. We have operated in 2 cases of ruptured papillary muscle. Neither of these were secondary to myocardial infarction; one being due to trauma, the other a complication of bacterial endocarditis. In both of these patients the papillary muscle could be repaired. Perhaps in the future, cardiac surgeons may adopt a more aggressive attitude toward these complications of acute myocardial infarction.

DR. ALFRED GOLDMAN, Beverly Hills, Calif.—I rise only to mention another type of ventricular aneurysm; that is one in which one cannot get around its base. We have had two such cases, and each time an attempt was made to clamp this very leathery dilated wall of the left ventricle, the blood pressure would drop. In each of these cases we made a repair after Beck's method, using a flap of pericardial fat pad and pericardium over the small weak spots that were encountered on the surface of the aneurysm. Both these patients are now about 2 years postoperative and neither has had any further coronary occlusion. They are both somewhat disabled cardiac-wise, but they are both alive and able to do light work.

I want to mention one test that might be valuable in determining the extent of a through-and-through infarct such as is present with this type of aneurysmal dilatation of the left ventricle. If the surface of the heart is explored with the unipolar electrode, using Prinzmental's technique there will be over any active myocardium, with pressure, an injury current measured by an elevation of the S-T segment. Over the surface of this kind of ventricular aneurysm, where there is no active muscle, Q waves occur without elevation of S-T segment on pressure with the electrode. One can demarcate in this way the edges of the aneurysmal, dilatation because pressure will produce no rise of the S-T segment over the through-and-through infarct replaced by fibrous tissue.

DR CLAUDE S BECK, Cleveland, Ohio —Not all aneurysms of the heart are saccular, and not all of them need to be excised I have had 2 patients with aneurysm large enough to have a paradoxical pulsation These were approximately 4 cm in diameter and bulged out approximately 2 cm A graft of fascia lata was placed over the aneurysm in one and in the other a graft of nylon was used These grafts were sutured to the adherent pericardium in the periphery of the aneurysm The graft was made tight enough to do away with the paradoxical pulsation and the outward bulge This method of treatment is recommended for aneurysms with a paradoxical pulsation and without a narrow neck In patients upon whom I operated for coronary artery disease, the incidence of aneurysm was 7 per cent The only aneurysms that need a supporting graft are those that definitely bulge out from the heart or have a paradoxical pulsation

DR CHARLES P BAILEY, Philadelphia Pa (Closing) —I want to thank all the discussors and I will try to answer the questions they have brought out There is little to say about Dr Bell's beautiful case, and we can only congratulate him upon his success Dr Mangiardi raised the question of why there was no remaining muscle tissue in the section of the aneurysmal wall That is, of course, the end result of an infarction that was through and through Some aneurysms do have muscle fibers in their wall, and they are the ones that occur after incomplete or partial thickness involvements Those that occur after secondary infarction of a previously infarcted area tend to be purely fibrous I do not know why Dr Mangiardi's patient lost his hypertension, but it would be a wonderful thing if we could only control this effect upon our patients

It is important to realize that even though there be muscular elements in the wall of an aneurysmal pouch, as long as you do not get beyond the limits or confines of the scar you will have good strong tissue to hold the sutures something you do not have in the experimental animal Therefore in the human patient you will seldom need Ivalon sponge reinforcement of your suture line

Dr Bosher's case is indeed fascinating, I suppose none of us has ever heard of anything like that before, and he has had a fine result Dr Jones, I think, is somewhat more fearful than he need be about clots within the aneurysm If you are gentle you can palpate the clot and not dislodge it I think it is important to know whether or not one of the papillary muscles is involved in the aneurysm, because if you are going to cut away its normal site of attachment you must be particularly careful to incorporate the stump within the suture line in the ventricular wall, lest you produce a severe degree of mitral insufficiency I think there may be many other ways than ours of taking off an aneurysm and I have no doubt Dr Jones' method will work In fact, about a year prior to our first case, Dr Glover, then my associate, did remove a ventricular aneurysm using a purse-string suture technique The patient did not survive but this probably was not related to the technique, there was an abdominal complication We think that if Dr Jones would get one of these special clamps which are available from the Pilling Company, he would not have to worry about breaking it

Dr Cooley's fascinating case of repair of the acutely perforated ventricular septum, of course, clearly shows the surgical possibilities in chronic aneurysm of the septum That is not a very common form of ventricular aneurysm but it does occur, and I cannot imagine any way of treating it except by the use of cardiopulmonary bypass and an open technique As a matter of fact, Dr Lillehei has already removed a ventricular aneurysm by an open technique

CLINICAL EVALUATION OF THE SURGICAL TREATMENT OF THIRTY-TWO CASES OF CORONARY ARTERY DISEASE

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THE ultimate objective in the approach to the problem of arteriosclerosis of the coronary arteries is prevention. Since a reliable method of prevention has not yet been developed, attention must be directed toward overcoming the effects of occlusive narrowing of the coronary arteries. Surgical revascularization of the heart provides a means of improving coronary circulation.

Schlesinger and Zoll[1] have shown that only the larger coronary arteries are affected by arteriosclerosis. The smaller arteries, and the veins and capillaries are not involved. These vessels, therefore, do not offer an obstruction to coronary blood flow. If oxygenated blood could be delivered to the uninvolved vessels, and the blood were equitably distributed throughout the myocardium, it is conceivable that the effects of diminished coronary inflow could be overcome. The validity of this theory has been demonstrated in the animal laboratory. Beck has shown that the mortality and the size of an infarct resulting from sudden occlusion of a major coronary artery can be significantly reduced in dogs following the Beck I[2] and Beck II[3] operations. These operations also produce a significant increase in coronary backflow as demonstrated by the Mautz-Gregg backflow studies.[4]

The results of these experiments have led to the development of certain principles required of a successful revascularization procedure. These principles include: (1) the delivery of an additional quantity of oxygenated blood to the ischemic areas of the heart, (2) the utilization of existing anastomotic channels, and (3) the stimulation of new intercoronary communications to provide a more equitable distribution of oxygenated blood.

Unfortunately, translation of laboratory data to the human being cannot be done with certainty. Objective demonstration of the value of a revascularization procedure cannot be obtained in the human being at this time. Results, therefore, must be evaluated on the basis of (1) amelioration of symptoms, (2) ability to return to work, and (3) length of survival. Proof of the feasibility of surgical revascularization of the heart must depend upon evaluation of a large number of cases closely observed for a long period of time.

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SELECTION OF CASES FOR SURGERY

As an aid in the selection of cases for surgery the status of the patient with coronary artery disease was classified according to the following categories:

1. Asymptomatic. Those patients who have had previous myocardial infarction but who no longer have symptoms. It was assumed that collateral circulation developed to such a degree as to overcome the effects of coronary occlusion and surgery was not indicated.

2. Progressive. Those cases in which the collateral circulation is unable to keep pace with a rapidly advancing disease process. Existing collateral circulation was considered to be inadequate for the patient to withstand surgery.

3. Stabilized. Patients with angina pectoris that is stabilized with regard to frequency and severity of attacks. This type of individual represents the ideal candidate for surgery.

4. Far Advanced. Those patients who have severe coronary artery disease with advanced myocardial degeneration and replacement fibrosis. Obviously, surgery can be of little value in this group since revascularization cannot alter the irreversible myocardial changes.

In the more specific selection of cases for surgery the following considerations arose:

1. Age. Age per se was not a factor in the selection of cases for surgery except for the Beck II operation and combined procedures. Physiologic age was considered to be of greater importance than chronological age.

2. Sex. Sex was not an important factor in the selection of candidates for surgery.

3. Time of Surgery. Surgery was not performed for at least six months after coronary occlusion. This time interval was fixed arbitrarily to ascertain the degree of anastomotic circulation that may have developed after infarction. If sufficient collateral circulation had developed, surgery was not deemed necessary.

4. Angina Pectoris. Angina of effort represented the prime indication for surgery. It is important to emphasize that the angina had to be stabilized with regard to frequency and severity of attacks. Status anginosus and angina decubitus contraindicated surgery.

5. Hypertension. Patients with severe degrees of hypertension were not accepted for surgery. A mild or moderate elevation of blood pressure without evidence of systemic degeneration was not considered a contraindication to surgery. Blood pressure was an important factor in the selection of patients for the Beck II operation and combined procedures.

6. Heart Size and Congestive Failure. The ideal candidate should not have evidence of cardiac enlargement or congestive failure. A slight degree of enlargement and mild congestive failure were accept Patien

more than minimal cardiac enlargement and evidence of persistent or progressive cardiac decompensation have advanced myocardial degeneration and fibrosis and cannot be aided by revascularization procedures.

7. Disturbances in Rhythm: Surgery was considered to be hazardous in the presence of arrhythmias since they are generally associated with extensive myocardial damage.

8. Associated Conditions: Advanced medical diseases such as generalized arteriosclerosis, pulmonary disease, hepatic or renal failure contraindicated surgery.

PREOPERATIVE AND POSTOPERATIVE CARE

All patients selected for surgery were admitted to the hospital one week prior to operation. Laboratory examinations included a standard 12-lead electrocardiogram, x-ray and fluoroscopic examination of the chest in the posteroanterior, left anterior oblique and right anterior oblique views, complete blood count, sedimentation rate, nonprotein nitrogen, urinalysis, and serologic test for syphilis. It has been our practice in the past three years to digitalize completely all patients routinely prior to surgery. Mercurial diuretics were administered if there was any suspicion of cardiac failure.

Postoperatively, the patient was placed in an oxygen tent for twenty-four hours. A pleural drainage tube was attached to a water seal and removed twenty-four hours after drainage stopped. Digitalis was continued postoperatively. Bronchoscopic examination and removal of bronchial secretions were performed as necessary. An electrocardiogram was obtained on the second postoperative day and at the time of discharge. A portable film of the chest was taken on the first postoperative day and films comparable with preoperative x-rays were obtained prior to discharge from the hospital. Narcotics were administered for the relief of pain. Blood transfusions and intravenous fluids were used only when indicated. If necessary, blood pressure was maintained by the slow, continuous, intravenous drip of Levophed. Antibiotics were administered routinely, postoperatively. Skin sutures were removed on the tenth postoperative day at which time the patient was permitted out of bed. Physical activity was restricted for the first month and then increased gradually until the end of the second month when full activity was resumed. Digitalis was continued until the end of the second month postoperatively. Nitroglycerine, gr. $\frac{1}{400}$ every three hours, was administered during the first three postoperative months. The patient was permitted to return to work between the fourth and the sixth month.

RESULTS

This series includes 32 patients with coronary artery disease operated upon during the past five years (Table I).

Age.—The youngest patient was 32 and the oldest was 64 years of age at the time of surgery. The average age of the entire group was 44 years.

Sex.—Only 3 of the 32 patients were females. No attempt was made to exclude a patient from surgery solely on the basis of sex. The higher incidence of coronary artery disease in the male and his desire to seek rehabilitation were the chief reasons accounting for the sex differential.

Angina Pectoris.—All patients subjected to surgery had angina pectoris. Nineteen of the 32 patients had to discontinue employment. The remaining 13 patients had to change to employment which required less physical and emotional effort or continue their occupation despite varying degrees of disability. The determining factors in the selection of cases for surgery were the frequency and severity of anginal attacks, and the degree of incapacitation. Of the 32 patients, 28 had moderate to severe angina with attacks appearing on walking three city blocks or less. In the remaining cases it was less severe. In 18 of the 32 cases, angina had been present for two years or more.

Of the 29 patients who were followed up to five years postoperatively, 24 or 82.7 per cent had an excellent result. They are completely free of angina and have been able to return to their former occupations. In one of the remaining cases the patient continues to have angina, but of much less frequency and severity than before operation. He has been able to return to part-time work. There were 2 fair results. These occurred in patients who had an omentopexy operation in addition to the Beck I operation. Two patients had a poor result. One died eighteen months after a Vineberg operation. The other has had repeated angina and several episodes of myocardial infarction and is still unable to resume his former occupation.

The most dramatic results were obtained following a successful Beck II operation.

Previous Myocardial Infarction.—For the purpose of this study an attempt was made to accept for surgery only those patients who had definite evidence of coronary artery disease. Therefore, previous myocardial infarction was an important factor in the selection of patients for operation. A definite history of myocardial infarction was obtained in 28 of the 32 cases. Thirteen of the 28 patients had more than one infarction. Of the remaining ones, 2 had strong but not conclusive evidence of coronary occlusion. Two patients did not have a history of infarction.

No patient was operated on sooner than six months following coronary occlusion. The longest interval between infarction and surgery was seven years.

Cardiac Enlargement and Congestive Failure.—Definite left ventricular enlargement was present in 7 of the 32 cases. One of these patients had marked enlargement of the heart with an advanced degree of congestive failure. He died on the operating table. Of the remaining 6 patients, 5 had associated dyspnea on exertion. Five patients had exertional dyspnea without evidence of cardiac enlargement. One patient had paroxysmal nocturnal dyspnea. All patients were relieved of their dyspnea postoperatively, and in all cases the heart size returned to normal.

TABLE I. SUMMARY OF THE CLINICAL HISTORY AND OPERATIVE RESULTS OF

CASE NO.	NAME	SEX	AGE	PREOPERATIVE							
				ANGINA* THRESHOLD (BLOCKS)	ANGINA* DURATION	EMPLOYMENT	DYSPNEA	CONGESTIVE FAILURE	CARDIAC ENLARGEMENT	ECG	NO. O PREVIO MYO CARDI INFAR TION
BECK II											
1	J. C.	M	43	2	6 yr.	Discont.	Exertion	–	–	Old post.	3
2	L. O.	M	56	½	2 yr.	Discont.	–	–	L. vent.	Old ant.	3
3	J. T.	M	39	2	1 yr.	Discont.	–	–	–	Old post.	1
4	W. K.	M	39	10	2 yr.	Cont.	–	–	–	Old post.	1
5	W. L.	M	35	2	6 yr.	Cont.	Exertion	–	–	Old ant.-sept.	2
FIRST STAGE BECK II											
6	W. S.	M	53	½	5 yr.	Discont.	Exertion	+	L. vent.	Right bundle branch block; old post.	2
7	T. W.	M	38	2	6 mo.	Discont.	Nocturnal	–	–	Old post.	1
BECK I PLUS OMENTOPEXY											
8	W. K.	M	45	½	6 mo.	Discont.	–	–	–	Old ant.-sept.	1
9	B. M.	M	58	2	1 yr.	Discont.	–	–	–	Normal	None definit
10	J. K.	M	41	2	5 yr.	Restrict.	–	–	–	Old post.	1
BECK I PLUS VINEBERG											
11	E. C.	F	55	½	3 yr.	Discont.	–	–	–	Old post.	1
12	J. L.	M	44	3	1 yr.	Discont.	–	–	–	Old post.	History vague
13	D. C.	M	54	½	3 yr.	Discont.	Exertion	+++	Generalized	L. vent. hypertrophy; old ant.-sept.	3

(*For footnote, see page 74.*)

CASES OF CORONARY ARTERY DISEASE TREATED BY SURGICAL REVASCULARIZATION

	POSTOPERATIVE							
OPERATIVE AND POSTOPERATIVE COMPLICATIONS	ANGINA*	EMPLOYMENT	DYSPNEA	CONGESTIVE FAILURE	CARDIAC ENLARGEMENT	LONG TERM ECG	RESULTS	REMARKS
	None	Full	None	–	–	No ess change	Excell	
Pulmonary embolus after second stage	None	Full	–	–	None	No ess change	Excell	
	None	Full	–	–	–	No ess change	Excell	Severe congestive failure after 2nd stage, 3rd operation revealed large aorticosinus fistula, occlusion of sinus gave excellent result
Patient died of cardiac arrest immediately after completion of first stage								
Died, "blowout" of vascular graft on the 13th PO day after 1st stage, received anticoagulants *postoperatively*								
Postoperative empyema	Minimal	Full	None	None	None	No ess change	Excell	
Pulmonary infarction	None	Full	None	–	–	No ess change	Excell	
Hiccough, belching, abdominal pain and distention, thrombophlebitis	None	Full	None	–	–	No ess change	Excell	Abdominal and lower intercostal chest pain but no angina, symptoms of diaphragmatic irritability persist
Hiccough, abdominal pain, gastric distress	5 blocks	Restrict	–	–	–	No ess change	Fair	
Hiccough, gastric distress, abdominal distention	5 blocks	Restrict	–	–	–	No ess change	Fair	Symptoms of diaphragmatic irritability
	None	Full	–	–	–	No ess change	Excell	
Thoracentesis, 350 c.c. serous fluid	None	Full	–	–	–	No ess change	Excell	
Died of cardiac arrest on operating table as internal mammary artery was being implanted into myocardium								Because of advanced myocardial fibrosis and advanced arteriosclerosis, the patient would not now be considered a candidate for surgery

				PREOPERATIVE						
				ANGINA*						
CASE NO.	NAME	SEX	AGE	THRESHOLD (BLOCKS)	DURATION	EMPLOY-MENT	DYSPNEA	CON-GESTIVE FAILURE	CARDIAC ENLARGE-MENT	ECG
VINEBERG										
14	S. F.	M	55	½	7 yr.	Discont.	Exertion	–	–	Old ant.
BECK I										
15	A. H.	M	32	2	1½ yr.	Discont.	–	–	–	Old post.
16	F. S.	M	64	4	15 mo.	Discont.	–	–	–	Old post.
17	G. J.	F	53	3	15 mo.	Discont.	–	–	–	Normal
18	J. T.	M	39	½	3 yr.	Restrict.	Exertion	–	L. vent.	Old ant.
19	W. C.	M	41	2	6 mo.	Discont.	–	–	–	Old ant.-sept.
20	V. S.	M	57	½	6 mo.	Discont.	Exertion	–	–	Old post.
21	J. C.	M	45	5	3 yr.	Restrict.	–	–	–	Normal
22†	W. C.	M	44	2	2 yr.	Discont.	Exertion	–	L. vent.	Old ant.-sept.
23†	S. S.	M	50	½	2 yr.	Discont.	Exertion	–	L. vent.	Right bundle branch block; old ant.
24	D. M.	M	44	2	1 yr.	Restrict.	Exertion	+	L. vent.	Normal
25	R. A.	M	34	2	1 yr.	Restrict.	–	–	–	Old post.
26	T. E.	M	41	1½	3 yr.	Restrict.	–	–	–	Old post.
27†	A. A.	M	38	2	3 yr.	Restrict.	–	–	–	Old post.
28†	N. R.	M	40	½	2 yr.	Restrict.	Exertion	–	–	Old ant.
29†	J. K.	M	43	5	1 yr.	Restrict.	–	–	–	Old post.
30†	H. G.	M	48	1	8 mo.	Discont.	–	–	–	Old post.
31†	M. C.	F	53	3	2 yr.	Restrict.	–	–	–	Old ant.-sept.
32†	J. B.	M	54	½	3 yr.	Restrict.	–	–	–	Old ant.-sept.

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*Measured as ability to walk city blocks.

†Cases 22, 23, 27-32 had first stage Beck II operation. Thrombosis of the vasc graft was noted at the time of the second stage operation. A Beck I procedure was formed at the time of the second operation.

I—CONT'D

OPERATIVE AND POSTOPERATIVE COMPLICATIONS	POSTOPERATIVE							
	ANGINA*	EMPLOYMENT	DYSPNEA	CONGESTIVE FAILURE	CARDIAC ENLARGEMENT	LONG TERM ECG	RESULTS	REMARKS
	½ block	Discont.	None	–	–	No ess change	Poor	Died 18 mo after operation
Operative and postoperative sinus tachycardia	None	Full	–	–	–	Improved T waves	Excell	
Acute interoseptal infarction on the 5th PO day	None	Full	–	–	–	No ess change	Excell	
Cardiac arrest on the operating table, hypotension until 5th PO day, severe angina on 19th PO day	None	Full	–	–	–	No ess change	Excell	
Sinus tachycardia for 13 days postoperatively	None	Full	None	–	None	No ess change	Excell	
	None	Full	–	–	–	No ess change	Excell	
Ventricular tachycardia during anesthesia, surgery 2 mo later, developed ventricular tachycardia during abrasion of epicardium	None	Full	None	–	–	No ess change	Excell	
Bronchopneumonia, mild	None	Full	–	–	–	No ess change	Excell	
Hemothorax on 8th PO day after first stage Beck II operation	5 blocks	Restrict	None	–	None	No ess change	Good	
	None	Full	None	–	None	No ess change	Excell	
	None	Full	None	–	None	No ess change	Excell	
	None	Full	–	–	–	No ess change	Excell	
	None	Full	–	–	–	No ess change	Excell	
	2 blocks	Restrict	–	–	–	Repeated myocardial infarcts	Poor	Relief of symptoms for 18 mo angina then returned, has had 3 coronary occlusions since surgery
	None	Full	None	–	–	No ess change	Excell	
	None	Full	–	–	–	No ess change	Excell	
	Minimal	Full	–	–	–	No ess change	Excell	
Postoperative atelectasis	None	Full	–	–	–	No ess change	Excell	
	None	Full	–	–	–	No ess change	Excell	

Hypertension.—Patients with more than a mild or moderate degree of hypertension were not considered for surgery. Only one patient (Case No. 19) had a past history of hypertension, and he was normotensive at the time of surgery.

Operative Complication.—The most common operative complication was the appearance of cardiac arrhythmias during the induction of anesthesia and during surgery. Although many types of arrhythmia were observed the most frequently encountered were nodal extrasystoles, nodal rhythm, interference dissociation, and auricular and ventricular premature contractions. These abnormalities appearing early in the operation were not considered to be hazardous and usually subsided with either adequate oxygenation or temporary cessation of the operative procedure. Ventricular extrasystoles uniformly appeared during abrasion of the epicardium and during procedures involving manipulation of the ventricles.

There were four instances of serious cardiac arrthyhmia. Cardiac arrest occurred in 3 patients. Two of these died; one on the operating table and the other on returning to his room. The third patient is alive and free of symptoms. In one case ventricular tachycardia occurred during the induction of anesthesia. Surgery was canceled and the tachycardia reverted spontaneously. When surgery was performed two months later, ventricular tachycardia appeared during abrasion of the epicardium. Intravenous procaine amide controlled the rhythm and the patient had an uneventful postoperative course.

Operative Mortality.—There were three operative deaths among the 32 patients, giving an operative mortality of 9.4 per cent. Two of the deaths were in the younger age group and were among the first 6 patients subjected to surgery. One patient died of cardiac arrest at the conclusion of the first stage of the Beck II operation. The second patient had a "blowout" of the aorticosinus graft on the thirteenth postoperative day. This was the only patient who received anticoagulants postoperatively. The third patient died of cardiac arrest during a combined Beck I and Vineberg operation. Left bundle branch block preceded the appearance of cardiac standstill and appeared as the internal mammary artery was being implanted in the ventricular myocardium. In the light of our present concepts, this patient would not now be considered a candidate for surgery since he had evidence of generalized arteriosclerosis, congestive failure, and marked cardiac enlargement.

There were no deaths among those patients undergoing the Beck I operation.

Postoperative Complications.—In all cases special care and attention had to be directed to the respiratory system. Retention of bronchial secretions with subsequent pneumonitis and localized areas of atelectasis were frequent, but not alarming, postoperative complications. In one case, bronchopneumonia was of sufficient magnitude to retard a smooth postoperative course. Significant atelectasis developed in one patient, and empyema occurred in one. Although small quantities of pleural fluid developed in almost all cases, a thoracentesis was required on only two occasions. In one of these hemothorax was present. Postoperative pulmonary infarction occurred in 2 cases. Gastric symptoms and signs of diaphragmatic irritability were noted after omentopexy.

One patient had postoperative hypotension of such a degree that Levophed had to be administered by continuous intravenous drip until the fifth postoperative day. Two patients developed sinus tachycardia. In one, it was mild and transitory. In the other, it persisted until the thirteenth postoperative day. In neither case was there any serious consequence of the tachycardia.

All patients but 2 were relieved of their angina during the immediate postoperative period. One patient, who had postoperative angina, developed an acute anterior myocardial infarction on the fifth postoperative day. He recovered and has returned to full-time work without disability. The other patient had severe angina on the nineteenth postoperative day.

Signs of pericarditis, as indicated by the appearance of either a friction rub, chest pain, or electrocardiographic changes were observed in all cases. The friction rub was audible almost immediately after surgery and persisted until the fourth or fifth postoperative day. In no case was there significant pericardial effusion or evidence of constrictive pericarditis.

In 8 (Cases 22, 23, 27-32) of the 15 patients who underwent the Beck II operation, thrombosis of the vascular graft was found at the time of the second-stage operation. A Beck I procedure then was performed in these cases. Two patients who submitted to the first stage of the Beck I operation refused the second stage. In only 3 cases was the graft known to be patent.

Electrocardiogram.—An abnormal electrocardiogram, including definite evidence of previous myocardial infarction, was obtained in 28 of the 32 cases. Two patients had right bundle branch block. There were no cases of left bundle branch block. All patients had a regular sinus rhythm. Electrocardiograms taken on the second postoperative day showed either no change in comparison with preoperative tracings or evidence of pericarditis. The changes attributed to pericarditis usually disappeared by the end of the first month. In general, late postoperative graphs were essentially unchanged over preoperative tracings.

Long-Term Deaths.—Of the 29 patients who survived surgery and who have been followed up to five years, only one has died since surgery. This patient had severe coronary artery disease with persistent angina. He died eighteen months after a Vineberg operation.

DISCUSSION

Two types of death result from coronary artery disease. The first is due to the appearance of ventricular arrhythmias. The second is the result of progressive myocardial degeneration. The former accounts for 90 per cent of deaths due to coronary artery disease. In many of these cases the actual pathologic damage to the heart is usually slight, and it would appear that the heart could still function adequately if only the rhythm could be protected. Arrhythmias appear when areas of unequal oxygenation of the myocardium are adjacent to one another. Harris and Rojas[5] demonstrated that ventricular extrasystoles arise from this area. Brofman and his associates[6] have been able to demonstrate that unequal oxygenation of the heart can result in ventricular fibrillation and death. Protection is dependent, in part, upon an equitable distribution of oxygenated blood throughout the myocardium. The intercoronary channels are an important mechanism of protection. They may develop naturally during the course of coronary artery disease. Failure of these anastomoses to develop can result in sudden death or progressive myocardial degeneration. One of the major objectives of surgical revascularization of the heart is to promote increased utilization of existing intercoronary channels and to stimulate the development of new anastomotic communications that will provide a more equitable distribution of oxygenated blood, and thereby protect the heart from the serious effects of coronary artery disease.

It must be realized that revascularization procedures do not stop or revert the progress of the vascular arteriosclerotic process. Dead muscle cannot be revitalized or replaced. Consequently, surgical revascularization cannot be applied to all patients having coronary artery disease. Those patients in whom the disease process has progressed to such a degree as to cause irreversible myocardial degeneration, or those patients in whom revascularization of the heart cannot keep pace with a rapidly advancing disease process, will not benefit from surgery. Those patients who present evidence of moderately advanced coronary artery disease as manifested by a slight degree of cardiac enlargement and minimal congestive failure in the presence of stabilized angina of effort represent borderline cases. As was observed in this series, the results are usually beneficial but a good result cannot always be expected.

The operative mortality for surgical revascularization of the heart is not unduly high, considering the nature of the disease process. The over-all operative mortality in this series was 9.4 per cent. Two of the deaths occurred in the early years of the study. There was only one death among the last 26 cases, resulting in a mortality rate of 3.8 per cent. A careful and intelligent choice of candidates for surgery and the selection of the proper surgical procedure for the individual case will lower the operative mortality and increase the number of patients benefited by revascularization of the heart. In spite of these precautions, and because of the seriousness of the disease process, it is unlikely that the operative mortality will be reduced below a certain minimal level.

Postoperative complications were not excessive. Careful attention to the physiology of the respiratory system can alleviate greatly the frequency and severity of postoperative complications. The most common problems are those related to retained bronchial secretions with subsequent atelectasis and pneumonitis. Although surgery is performed on a diseased heart, the complications arising from the cardiovascular system are less than would be anticipated.

The results of surgery in this series of patients have been most gratifying. In over five years of postoperative observation, there has been only one long-term death. Of the surviving patients, 82.7 per cent have been almost completely relieved of angina and have been able to return to their former occupations. Two of the patients in this series have shown little improvement, but are in no worse condition than preoperatively. Only 2 of the patients in the series had a poor result.

Greater emphasis should be placed on the principles of coronary artery surgery rather than upon individual surgical techniques. Any operation that is designed to revascularize the heart should be tested in the animal laboratory before being applied to the human being. The Beck I operation fulfills the laboratory criteria for a successful surgical procedure. Clinical experience indicates that this operation permits a rather wide latitude in application. The operative mortality is low and the results of surgery have been favorable. Combined procedures are not recommended for routine use.

SUMMARY AND CONCLUSIONS

1. The results of the surgical treatment of coronary artery disease in 32 cases together with a five-year follow-up are presented.
2. Surgical revascularization of the heart in properly selected patients is an important and valuable method of treatment of coronary artery disease.
3. Laboratory experiments and clinical experience indicate that the Beck I operation, at present, is the procedure of choice in the surgical treatment of coronary artery disease.

The authors wish to extend their appreciation to the New Jersey State Department of Health for the cooperation and assistance received in the performance of this study.

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CASE OF GRANULAR-CELL MYOBLASTOMA (ORGANOID TYPE) INVOLVING ARM, LUNG, AND BRAIN, WITH TWENTY YEARS' SURVIVAL

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SURVIVAL of twenty years or more is rare in a case of malignant tumor, proved histologically, after extensive and multiple metastases.

INTRODUCTION

In 1953, a long survival with a *malignant melanoma* was reported by Galgano.[9] It was unusual because of the long time between the detection of the primary lesion and the subsequent first appearance of pulmonary metastases, and the unusually long time between the development of metastases in the lung and the death of the patient. The primary lesion was in the skin of the right temple and proved to be a melanoma histologically. It was not until eleven years later that any pulmonary symptoms developed showing, by x-ray examination, a small nodule in the left lung. There was an additional long interval of nine years between the detection of pulmonary metastases by x-ray examination and the death of the patient, in all, a period of forty years from the development of the original skin lesion until a fatal issue ensued. The patient finally died at the age of 86 (her first skin lesion having developed at the age of 46) of widespread pulmonary and other metastases.

In 1952, Ficarra[7] described a similar case (with post-mortem examination) of a patient, twenty years after a bilateral mastectomy for carcinoma. The author presented his case as an instance of so-called "tissue resistance" to cancer. There must be, he thought, some type of immunity or pseudo-immunity to cancer in certain fortunate individuals to account for the long course of disease occasionally seen in verified cases of malignancy. No matter what term is used, many clinicians have observed that of 2 patients with the same type of cancer, of approximately the same age, with the same duration of the primary disease, one patient may die within a year or less and the other may survive for decades. In the case reported by Ficarra, that of a 60-year-old woman, the patient had had a bilateral mastectomy for carcinoma twenty years previously and had not received any x-ray therapy postoperatively. She had carried out her usual family duties, her health had been good for almost twenty years after the bilateral mastectomy until several months before her death when she developed loss of weight and a nonproductive cough. One month before her

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hospitalization, ascites developed. At autopsy, cancer nodules were found from head to knees and at both elbows. No metastases were found in the extremities beyond the elbows or knees. The skeleton and the viscera were studded with adenocarcinoma. One of the photographs shows the omentum which was literally riddled with metastatic cancer nodules. The author explains the absence of metastases below the elbows and knees by the investigations of Batson who, in 1940, showed the function of the vertebral veins and their importance in the spread of metastases. These veins extend from the coccyx to the skull and communicate with the veins in the ribs, shoulder girdle, pelvic girdle, upper end of the humerus and femur. In this case, the lesion was the usual variety of adenocarcinoma of the breast. It was not even known, the operations having been carried out in a distant country, whether *each* breast was involved originally in carcinoma.

Hutcheson[11] reported, in 1952, a case of metastatic cyst adenocarcinoma of the ovary thirty-three years after removal of the primary growth. This is also an extreme example of the ability of malignant cells to remain dormant for long periods and then to proliferate again. This particular tumor caused death thirty-three years after removal of the primary pelvic growth without having given rise to symptoms during the long interim. Recurrence, verified at post-mortem examination, in the pelvis and lower abdomen was detected only six months before death. Edema of the lower extremities then developed. The patient had been entirely well from the time of the original operation and tumor removal thirty-three years previously until the terminal edema. A complete necropsy showed no other primary tumor.

Reyes and Horrax[1] described (1950) a case of metastatic (brain) melanoma extirpated from the left frontal lobe, which was the size of a duck's egg, after previous melanomas had been removed from the breast and axilla in a 57-year-old man. The patient was alive, well, and working as an insurance salesman almost four years later. The *original* dark mole had been widely excised from the left shoulder two years before craniotomy.

These cases are examples of rather common types of malignancy, but the case to be reported, as the title of this paper suggests, is of *obscure histologic origin* and therefore of added interest for that reason alone.

CASE REPORT

C. H., a 35-year-old white man (referred by Dr. Arthur Brinkley, of Richmond, Va.), was admitted Aug. 9, 1952, for the first time to the Medical College of Virginia Hospital for treatment of presumably metastatic tumors of the left lung. Past history disclosed that in 1935, when the patient was 18, a mass was removed from the right biceps muscle in another hospital. After considerable microscopic study, the final diagnosis at that time was hemangio-endothelioma. In the spring of 1936, x-ray films of the chest revealed three nodules in the left lung and several questionable nodules in the right lung. The patient was given 500 r of x-ray therapy to the lungs from the spring of 1936 to the spring of 1937. Subsequent periodic chest x-ray examinations had continued to show a persistent shadow in the basilar region of the left lower lobe, and several rather questionable densities in the right lung field. There had been a slow, progressive enlargement in the left lower lobe lesion from 2 cm. in 1937 to 4 cm. in 1952 (Figs. 1 and 2).

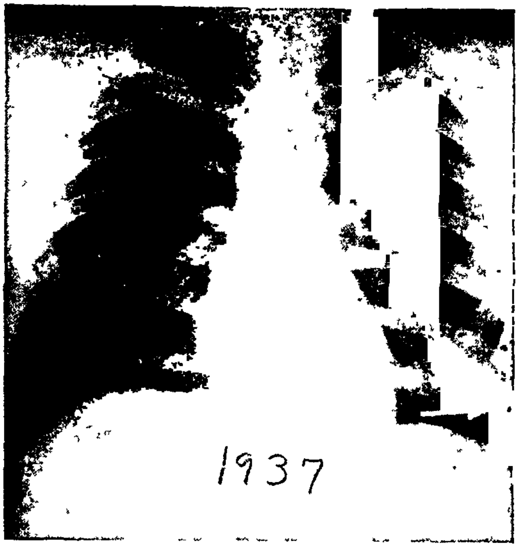


Fig 1.

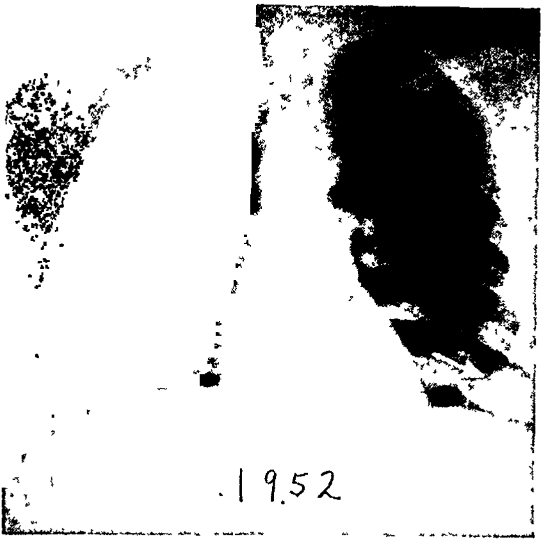


Fig. 2

Fig. 1.—Roentgenogram of chest made in 1937 which shows well-defined round density in left lower lobe (arrow).

Fig. 2—Roentgenogram of chest made in 1952 which shows increased size of density noted in 1937 film (arrow).

Fig. 3.—Ventriculogram showing distortion and compression of right lateral ventricle by intracerebral metastatic tumor, with shift of ventricular system to the left

The patient had remained asymptomatic during these years and had had no cough, weight loss, chest pain, or hemoptysis. There had been no evidence of local recurrence of the primary lesion. During the past two months, however, the patient had complained of daily frontal headaches and bilateral tinnitus, neither of which he had ever had before. He was seen by the neurosurgeons prior to his present admission and no objective evidence of intracranial metastasis or of increased intracranial pressure was found.

Physical examination revealed a temperature of 98.2° F., pulse 72, and respirations 16. The lungs were clear to percussion and auscultation with no rales present. Results of all other examinations were also negative.

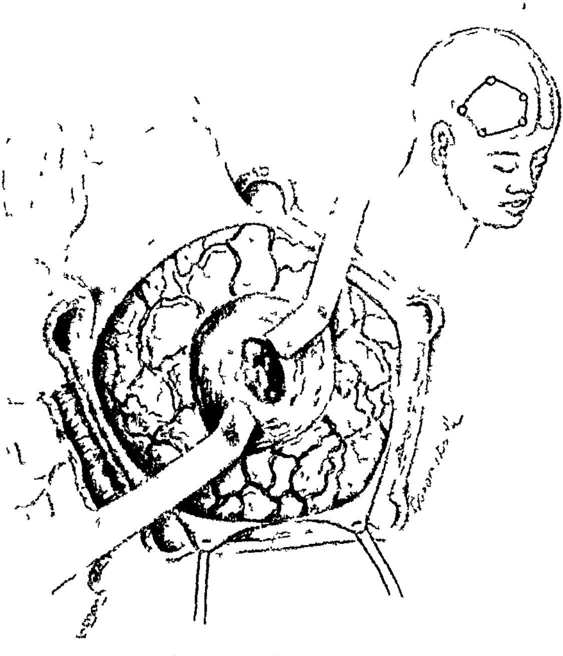


Fig. 4.—Artist's drawing showing location and appearance of single deep metastatic lesion in posterior aspect of right frontal lobe. The smaller figure shows the location of the bone flap.

Laboratory findings were noncontributory, except for the chest roentgenogram. This revealed a well rounded nodule in the region of the lateral basal segment of the left lower lobe. No definite nodules were seen in the right lung fields at this time.

On the third hospital day, a left thoracotomy was done. A large nodule measuring approximately 4 cm. in diameter was found in the lateral basal segment of the *left* lower lobe. There were two smaller nodules, each less than 1 cm. in diameter, in the anterior and posterior segments of the left upper lobe, respectively. A basal segmental resection and wedge resection of the smaller nodules in the upper lobe were done.

The patient was discharged Aug. 23, 1952, but two days prior to discharge, a planogram of the *right chest* revealed an area of density 2 cm. from the posterior wall which suggested a metastatic nodule. The patient was to return in three weeks for an exploratory thoracotomy on the right side.

However, during the interval at home, the headaches which the patient had experienced prior to chest surgery returned (one week following discharge). These were more severe than previously and were confined to the frontal and occipital regions. There were no localizing neurological symptoms. The patient was hospitalized again at the Medical College of Virginia Hospital on Oct. 2, 1952, for further study.

Physical examination then revealed a bilateral papilledema of three diopters. The reflexes were slightly more active on the left side. A craniotomy was performed on Oct. 9, 1952, for a "probable metastatic lesion in the right frontal lobe," the ventriculogram having suggested such a lesion (Fig. 3) earlier on the same day.

After opening the dura, a soft area was evident in the right posterior frontal region. A circular wedge-shaped portion of the cerebral cortex and subjacent white matter was removed down to and overlying a firm, well-circumscribed, small tumor nodule, about 2 cm. in diameter (Fig. 4). The mass was enucleated in its entirety. The bone flap was discarded because of cerebral edema. Portions of the tumor were sent in a sterile container to the tissue culture laboratory at the Columbia-Presbyterian Medical Center, New York City (see below). The remainder was utilized for pathologic examination.

The postoperative course was entirely satisfactory and the patient was discharged twelve days after the operation, to be followed as an outpatient. A thoracotomy on the right side was still contemplated.

After a tantalum plate insertion in the skull (December, 1952), the patient having remained symptomatically free, the right lung was explored on Jan. 22, 1953. The entire right lung was carefully palpated and a very small nodule (2 to 3 mm. in diameter) was found and removed from the inferior margin of the middle lobe. This was microscopically identical with the left pulmonary lesion removed in August, 1952.

In 1954, he developed definite signs of increased intracranial pressure and it was obvious that he had multiple intracranial metastases for a year or more before death. The patient was then given x-ray treatment to the brain: April to August, 1954 (3,000 r—tumor dose). A high degree of choked discs developed bilaterally and he became blind several weeks before he died at his home on June 3, 1955. There were several external discrete nontender but firm swellings on the scalp at the time of death. A post-mortem examination unfortunately was not obtainable.

Pathologic Findings.—

We were fortunate in having available a slide of the lesion removed from the forearm in 1935. This showed alveolar groupings of tumor cells, the groups being separated only by thin-walled, vascular spaces (Figs. 5 and 6). The cells of the groups were large, but fairly uniform with large, bizarre nuclei and prominent nucleoli. A pseudoglandular pattern was sometimes distinguished, but this was thought to be artifactual.

1. *Lung lesions, August, 1952:*

Gross (S-52 5371): The specimen consisted of the basal segment of a lung 11 by 13 by 9 cm. and weighing 99 Gm. The pleura was smooth, and a mass was palpable in the region of the diaphragmatic pleura. On cut section, a well-circumscribed tumor 3 by 2.7 cm. was found (Fig. 7). This had a gray-pink color, a fibrous texture, and showed focal hemorrhage. The bronchus leading toward the tumor was normal. In addition, there were three different portions of tissue. The first was from the upper lobe, measured 2.5 by 2 cm. and showed, on cut surface, a sharply circumscribed nodule 6 by 2 mm. The second specimen, also from the upper lobe, measured 2.5 by 0.3 cm., and contained a similar nodule 2 mm. in diameter. The final tissue was a peribronchial lymph node 1.8 by 1 cm. showing no unusual features.

Microscopic: Sections of the tumor mass and smaller nodules showed similar features. They were obviously metastatic lesions and almost identical with that noted in the lesion removed in 1935 (Figs. 8 and 9). Innumerable pseudo-alveoli rather closely

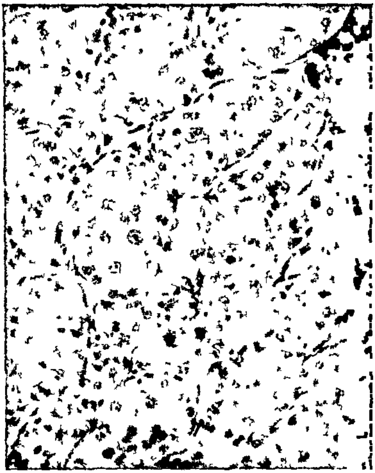


Fig 5

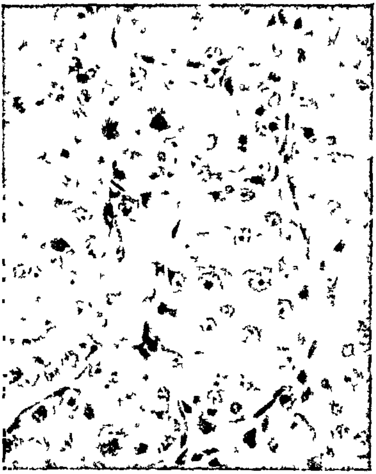


Fig 6

Fig 5.—Tumor from forearm to show pseudo-alveolar grouping of tumor cells in a highly organoid pattern

Fig 6.—High-power view of Fig 5 to show details of the individual tumor cells Coarse and fine granules are present in the cytoplasm

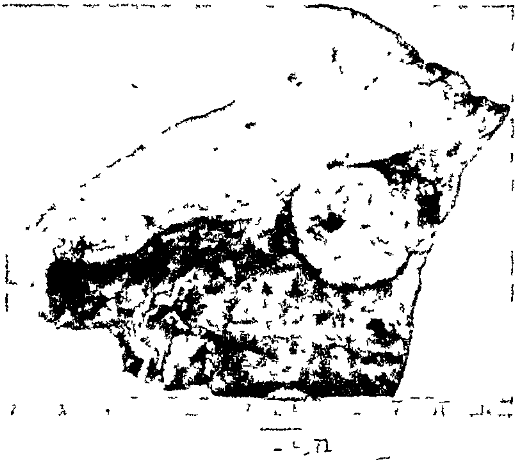
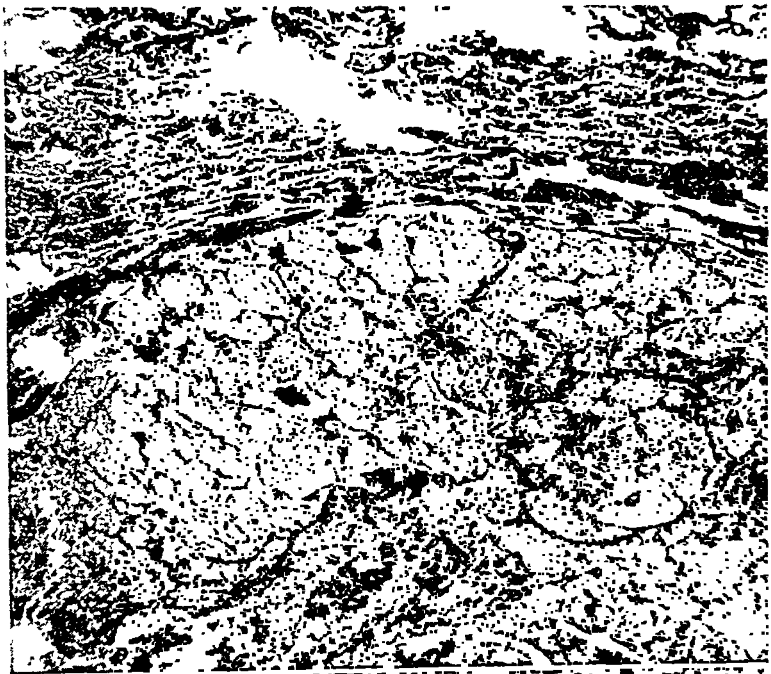


Fig 7.—Basal segment of left lower lobe of lung with well-defined nodule of metastatic tumor beneath the diaphragmatic pleura.

Fig. 8.



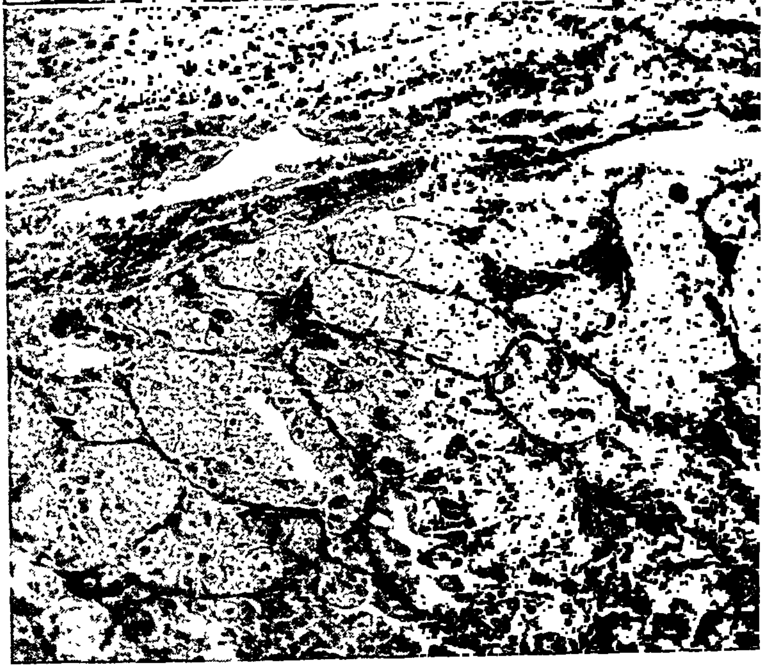


Fig. 9.

Fig. 8.—Photomicrograph of metastatic lung tumor to show similar organoid pattern to that of the primary growth.

Fig. 9.—Medium-power view of Fig. 8. Compare with Fig. 5.

placed were seen; these were made up of sheets of large, granular cells with rounded vesicular nuclei. The granularity of the cells varied, being fine and evenly distributed in some cells, while in others in was condensed into small, amorphous aggregates. Stains for mucin were negative, but a fat stain showed appreciable droplets of fat in some areas.

2. *Cerebral lesion, October, 1952:*

Gross (S 52 6667): The specimen consisted of a small, encapsulated, nearly spherical, firm, grayish-pink tumor, to which was attached bits of blood-stained brain tissue (Fig. 10). The tumor measured 1.8 by 1.5 by 1 cm. and weighed 3 Gm. The cut surface was homogeneous, magenta, firm and smooth, and surrounded by a capsule, 0.5 to 1 mm. in thickness.

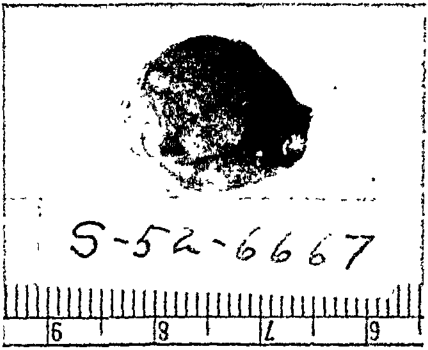


Fig. 10.—Hemisection of metastatic tumor from frontal lobe.

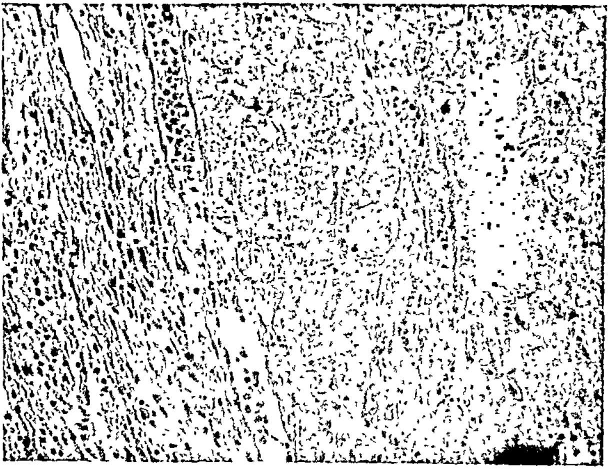


Fig. 11.—Metastatic brain tumor to show compression of brain tissue on [illegible] t. Note g[illegible] es in cytoplasm of tumor cells.

Microscopic: The features were similar to that noted above, and need no further elaboration. Despite the gross impression, encapsulation was rather poor (Fig. 11).

3. *Lung lesions, January, 1953:*

Gross (S-53 521): The specimen consisted of two small pieces of bluish-colored lung tissue from the right middle lobe, and a small white piece of tumor tissue 5 by 4 by 3 mm.

Microscopic: The same organoid pattern as noted in the previous surgical specimens was maintained.

Final Diagnosis.—Granular-cell myoblastoma, organoid type, of biceps muscle with metastasis to both lungs and right frontal lobe of brain.

DISCUSSION

This tumor is an interesting one about which there is some difference of opinion among pathologists as to its histogenesis. Three names have been used to describe it, namely, alveolar soft part sarcoma, malignant granular-cell myoblastoma of the organoid type, and malignant nonchromaffin paraganglioma.

Alveolar soft part sarcoma was favored by Christopherson, Foote, and Stewart,[3] and these authors reported 12 cases in 1952. Six of the patients developed metastases and, of these, 4 had died at the time of their report. The authors favored the term "alveolar soft part sarcoma" because it was descriptive in the histologic sense and noncommittal concerning its histogenesis.

Smetana and Scott[17] believed that the tumor, of which they studied 14 cases, was derived from nonchromaffin paraganglia. To support their thesis they cite the close relationship of these growths with nerves and their histologic similarity to carotid body tumors. In addition, the authors make reference to the finding by Lent Johnson of vascular glomera structurally similar to the carotid body in relation to the blood vessels of Hunter's canal.

Stout[18] took issue with the term of nonchromaffin paraganglioma. He did not entirely reject Smetana's hypothesis but still felt unconvinced since paragangliomas of the carotid body type almost never metastasize, while the tumor under discussion is regularly malignant. Stout's experience with the organoid variety of tumor comprised 11 cases studied in the laboratory of Surgical Pathology at Columbia University.

Willis[19] emphasized that he was uncertain as to the true nature of this tumor. He thought, in fact, that the term "myoblastoma" was being used as a "dumping ground" for all sorts of large granular-cell tumors, probably of *diverse* origins.

This paper does not propose to resolve the argument concerning the nature of this tumor. However, as mentioned previously, the cerebral metastatic material was submitted to Dr. Margaret Murray's tissue culture laboratory at Columbia University with the following final report from Dr. Murray:

> This tumor grew quickly and well, and throughout the 51 days that it was kept in vitro showed frequent, usually normal, mitoses. The tendency to proliferate may have been working against cell differentiation. The cells were of a general mesoblastic appearance and varied considerably in size. There was from time to time a bewildering variety of cell forms; but the most frequent, recurrent and recognizable specialization was in the direction of skeletal muscle: long, ribbon-shaped cells with abundant longitudinal striae were characteristic. These forms developed in vitro from the small, roundish,

> relatively compact cells which made up the explanted tumor nodules. Their nuclei also developed in vitro from being small and round, with 1 or 2 nucleoli, to being larger, oblong and vesicular, with several nucleoli. These changes are reminiscent of regenerating skeletal muscle, and of the behavior of adult skeletal muscle in vitro. Multinucleate cells, anastomosing ribbons, and large cells containing bizarre multilobate nuclei are also present. These too are consistent with the picture of skeletal muscle—both normal and neoplastic.
>
> This could be a granular cell myoblastoma. Apparently these tumors are rather protean in their manifestations in vitro—but most of the characteristic cell types seen in cultures of them are also characteristic of some form of skeletal muscle. This principle applies in this tumor. From its behavior in vitro, I do not see any grounds for aligning this tissue with the paraganglioma.[11]

Kelly and Langston[12] totaled the known cases of pulmonary resection for metastatic malignancy and found 109 instances with a two-year survival rate of 23.2 per cent. A number of long-term "cures" were reported, however. The results with sarcomas have been at least as satisfactory as with metastatic carcinoma.

Bilateral resection for metastatic pulmonary malignancy has been performed in only a few cases. Alexander and Haight's patient[1] who had bilateral pulmonary lobectomy after removal of a neurogenic sarcoma of the forearm was known to be well twelve years after the second lobectomy. Hood, McBurney, and Clagett[10] recorded a patient who had bilateral pulmonary resection for metastases eleven and fourteen years after excision of a fibrosarcoma of the leg. She was well forty months after the last operation. Churchill[4] mentioned a patient who had been treated with bilateral partial lobectomy. In addition to the patient reported here, we are familiar with 3 additional unreported cases of bilateral pulmonary resection for metastatic malignancy.[2, 5, 8]

Efforts to eradicate both pulmonary and cerebral malignant deposits are not likely to prove successful except in the very rare instance. This aggressive surgical approach could be expected to yield best results in the patient whose primary lesion is located in the lung. In the London Hospital autopsy records there were 85 cases of primary carcinoma of the lung associated with cerebral metastases. In 26 of the 85 cases, a solitary cerebral metastasis was demonstrated. Sporadic reports have appeared in the literature describing excision of apparently solitary cerebral metastases some time after pulmonary resection for primary lung tumor.

Reports of excision of both metastatic pulmonary and cerebral lesions are far more rare. Effler and Blades[6] reported a case of adenocarcinoma of the colon with resection of the primary lesion, subsequent pulmonary lobectomy for metastasis and finally excision of a cerebral metastasis two years after removal of the primary tumor.

A remarkable case was described by Moore, Perese, and Staubitz[13] in which removal of a testicle for teratocarcinoma was followed successively by craniotomy, pneumonectomy, two additional craniotomies and, finally, by excision of an isolated jejunal lesion. The patient was alive and well at the time of the report four years after the orchiectomy.

The accumulated experience in the treatment of multiple metastatic pulmonary malignancy is obviously rather meager. We are unaware of any previous attempt at bilateral pulmonary resection with excision of cerebral metastasis as well, such as was performed on our patient.[16]

SUMMARY

In summary, the case of a white man is presented who, at the age of 18, developed a soft tissue tumor in the right biceps muscle. This was excised and was followed 17 years later by surgically verified bilateral pulmonary and cerebral metastases with fatal issue almost three years after the removal of the latter lesions and twenty years after the original appearance and excision of the soft tissue muscle mass. In our opinion, the tumor was a granular-cell myoblastoma of the organoid type. This interpretation was in part supported by the results of tissue culture studies of the cerebral metastasis.

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SPONTANEOUS BILATERAL CHYLOPNEUMOTHORAX

A CASE REPORT

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SPONTANEOUS chylopneumothorax is a rare condition. We have been unable to find any reported case of spontaneous bilateral chylopneumothorax in the American or British literature and have found only one case of spontaneous chylopneumothorax which was unilateral.[1] This case, which followed lifting, was treated conservatively with recovery. It was postulated that the patient had ruptured a pulmonary bleb, developed a spontaneous pneumothorax, and had torn a lymphatic collateral, giving rise to the chylous fluid.

The following case of bilateral chylopneumothorax was found to be due to a diffuse pleural mesothelioma. Pleural mesothelioma is of infrequent occurrence and no report of a case with chylous fluid was found in a review of the American literature.

CASE REPORT

C. C. N., a 25-year-old white man, was admitted to Birmingham Veterans Administration Hospital on April 28, 1956, with a chief complaint of shortness of breath for one week and dyspnea for twenty-four hours. He stated that he had lost weight and strength for a year. Exertional shortness of breath had been present for four to six months. There was no pleural pain, no history of trauma, and the past history was negative.

The review of systems was not significant.

The patient's father had died of carcinoma of the stomach, and all of his father's family had died of cancer. There was no family history of tuberculosis.

Physical examination revealed a small, thin, white man in acute respiratory distress. Respirations were labored and there was slight intercostal retraction on inspiration on the right. Examination of the head, eyes, ears, nose, and throat revealed no abnormalities. There was no neck vein distention and no cervical lymph nodes were palpable. The trachea deviated slightly to the right. The entire right chest was dull to percussion and the left base also. Tactile fremitus was reduced and breath and voice sounds absent over these same areas. No friction rub was heard. Clubbing of the fingers, with cyanosis of the nail beds, was noted. The remainder of the physical examination was negative. Fluoroscopy revealed a fluid level up to the fourth intercostal space on the right. The initial impression of the ward physician was pleural effusion due to tuberculosis or lymphoma. Thoracentesis on the right yielded 1,400 c.c. of milky white fluid. The dyspnea decreased. A continuous air leak was present so a large gauge needle was left in the pleural space and attached to water-seal drainage. With suction, no re-expansion of the lung occurred. A surgical consultation was obtained and review of the initial chest x-ray revealed the presence of air and fluid in both pleural spaces with partial collapse of both lungs. Intercostal catheter suction on the right was instituted but did not re-expand the lung. The chylous fluid continued to drain in large amounts. Sudan III stain of the fluid indicated a high fat content. Cell studies on

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the fluid revealed large numbers of mononuclear white cells and individual mesothelial-type cells with no malignant characteristics. The patient continued to lose 2,000 to 3,000 c.c. of chyle daily. He maintained a good oral intake of fluid and solid foods.

More air began to accumulate in the left pleural space and dyspnea increased. Intercostal catheter drainage on the left removed 2 L. of chyle and much air. Ventilation improved. With clearing of the right pleural space of all fluid, enlargement of the right superior mediastinal shadow was seen on x-ray. Bronchoscopy was negative. Thoracotomy was performed to arrive at a definite diagnosis, to attempt to close the air leaks, decorticate and re-expand the right lung and, also, to control the debilitating chylothorax. A preoperative diagnosis of bilateral chylothorax of unknown etiology, possible mesothelial cell tumor, was made.

At operation on the right, all pleural surfaces were extensively replaced by tumor growing in sheets with some areas thickened up to form trabeculations. The lung was about 75 per cent collapsed, affecting all three lobes equally. The visceral pleura was replaced by a thin layer of this tissue which puckered the lung in areas. There were many minute air leaks, the source of the pneumothorax. The chyle appeared to rise from many areas over the mediastinal parietal surfaces and there was no intercommunication between the pleural spaces or localized disruption of the thoracic duct. Frozen section diagnosis of a malignant mesothelial cell growth was made. Although decortication was not successful, it was hoped that ligation of the thoracic duct would decrease the effusion of chyle. The thoracic duct was ligated just above the diaphragm and dissected proximally for 4 inches and excised. Three large thoracostomy tubes were left in the pleural space and attached to water-seal drainage bottles with suction. Complete re-expansion of the involved right lung could not be expected.

The pathologist reported, on permanent sections, malignant mesothelioma of the pleura, composed of uniform cells growing in sheets and strands, separated by a dense corrective tissue stroma. The cells showed vesicular nuclei, prominent nucleoli, and pale cytoplasm. In areas the cell boundaries were distinct. All tissue submitted showed the same type of infiltration.

The postoperative course was characterized by continued drainage of chylous fluid, and gradual deterioration in spite of a high caloric intake. Persistent air leaks, bilaterally, made it necessary to leave the thoracostomy tubes in both pleural spaces. Fractionated doses of nitrogen mustard in the pleural space did not decrease the amount of fluid draining daily. It continued to vary between 2,000 and 3,000 c.c. per day. Twenty days after thoracostomy, the patient quietly died.

Autopsy showed complete involvement of visceral and parietal pleura bilaterally with growth over the pericardium. The tumor extended into and below the diaphragm to involve the liver, the right adrenal, and the kidney. The mediastinum was extensively and deeply replaced by the growth.

DISCUSSION

The two uncommon features of the above case are: (1) the occurrence of spontaneous bilateral chylopneumothorax and, (2) the fact that it was due to a diffuse pleural mesothelioma.

Pleural mesothelioma will not be seen frequently in thoracic surgery and yet it is seen often enough to be kept in mind in the differential diagnosis of tumors of unknown types.[2] In a series of 60,042 necropsies, collected from various sources, pleural mesotheliomas were found in 43 cases or an autopsy percentage of 0.07 per cent. Stout[3] reported an accumulation of 72 cases in the laboratory of surgical pathology of Columbia University. Thirty-four were pleural and thirty-eight were peritoneal.

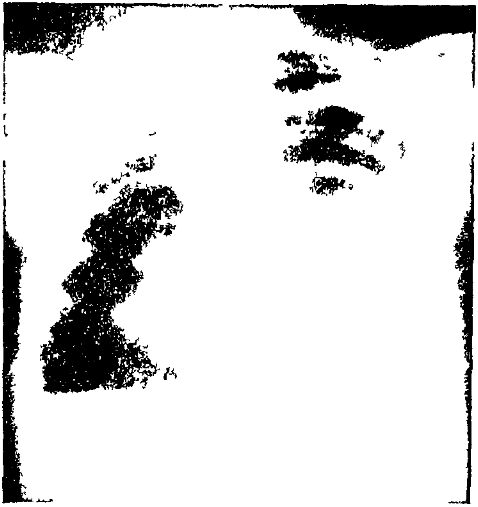

Fig 1 —Bilateral chylopneumothorax

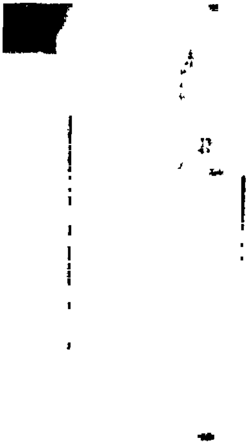

Fig 2 —Fluid aspirated from the right chest.

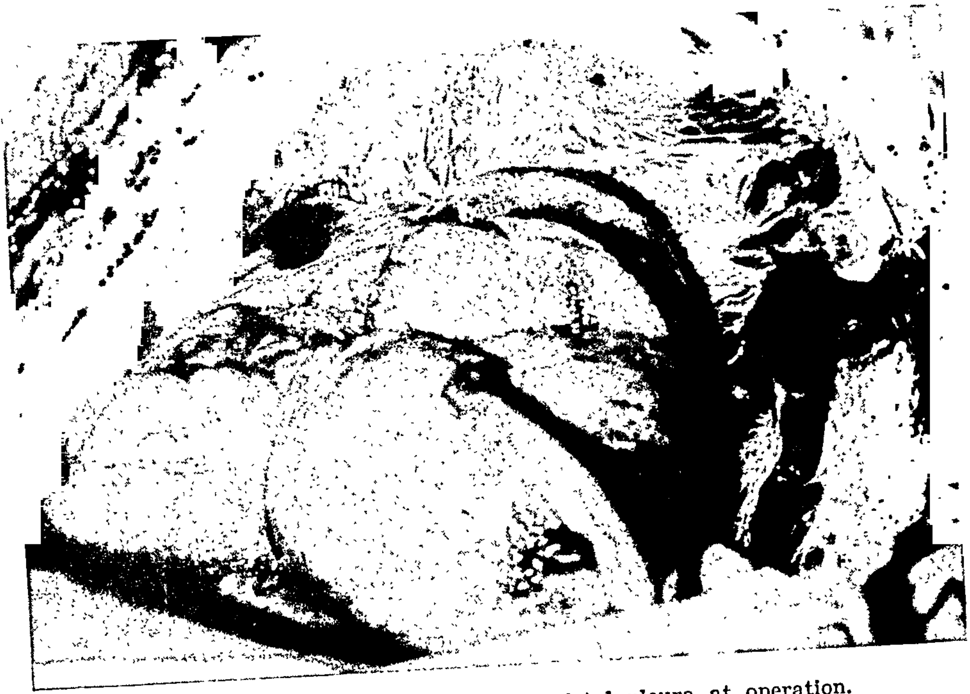

Fig. 3.—Tumor of visceral and parietal pleura at operation.

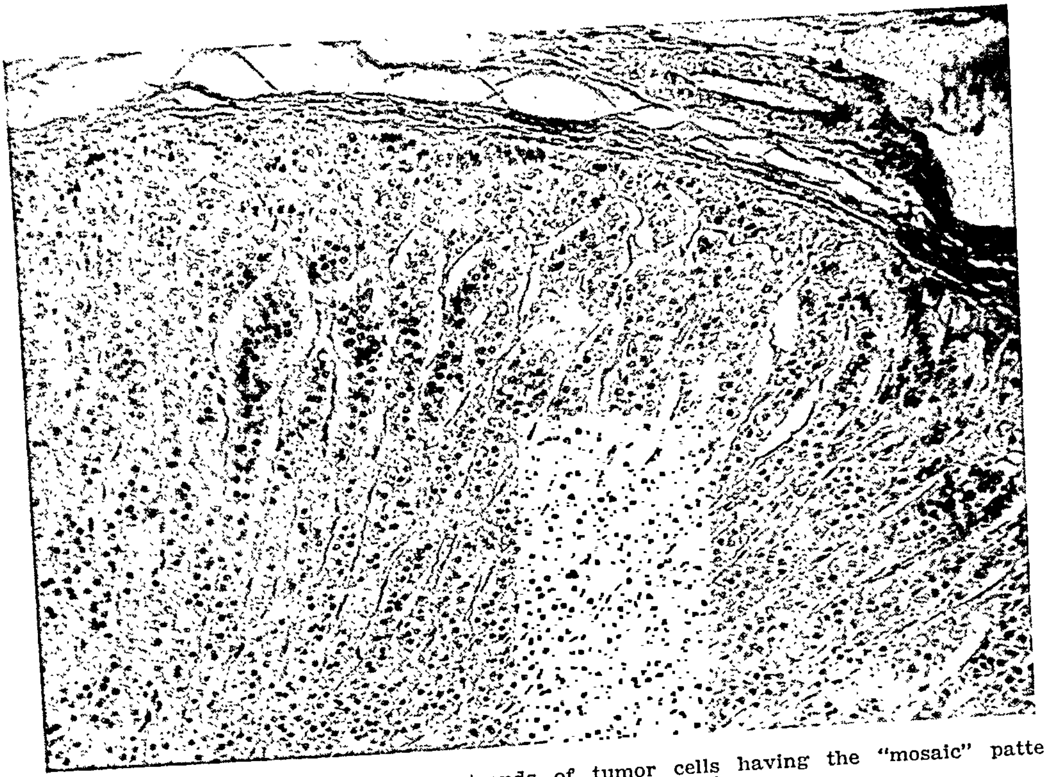

Fig. 4.—Microscopic picture showing strands of tumor cells having the "mosaic" pattern due to very distinct cell boundaries.

In 1950, Stout[4] reviewed the pathology of mesotheliomas and classified them in two main groups

1. The diffuse variety which is highly malignant and microscopically shows epithelial elements forming tubular structures These cells show the cultural characteristics of mesothelial cells and the ability to secrete hyaluronic acid.

2. Benign and malignant solitary fibrous mesotheliomas These occur both in the pleura and in the peritoneum. They lack epithelial elements and consist of a bizarre stroma with spindle cells predominating The benign type arises from a localized area of parietal or visceral pleural and projects freely out into the pleural space The malignant type invades adjacent lung parenchyma and does not project freely into the pleural space This variety has frequent mitosis, and variation in size and staining properties of the cells The stroma is sparse in reticulum and collagen with areas of hemorrhagic necrosis.

Pleural mesotheliomas usually appear in the age group from 40 to 60 years The ratio of males to females is approximately 2 to one. The symptoms and signs are variable and minimal, initially. This is an important feature of the clinical picture of both localized and diffuse mesotheliomas Chest pain of a mild and intermittent nature is the most common complaint and may be elicited only on questioning. The first signs usually are those of a pleural effusion which rapidly re-accumulates following thoracentesis It is usually bloody. Many times it is only after thoracentesis that a thickened pleura or a nodular density may be seen on x-ray. Any area of thickening along the mediastinal or lower pleural surfaces is suggestive. Pleural fluid cytology may reveal an increased number of mesothelial cells and many red blood cells

The diagnosis is usually not made until exploratory thoracotomy is done The histologic picture of mesothelioma is sufficiently characteristic to allow a certain diagnosis and this is of a practical importance in thoracic surgery, particularly concerning the localized type This type when excised has a better prognosis than bronchogenic carcinoma Therefore, it is important not to mistake it for an inoperable pleural metastasis from a bronchogenic carcinoma. There is no satisfactory treatment of the diffuse type. As far as can be determined every case reported and followed has been fatal

In the case just reported it must be postulated that the cause of the nontraumatic chylothorax was compression, invasion, or occlusion of the thoracic duct by the tumor growth at multiple sites This was not affected in the least by proximal ligation and excision of the thoracic duct It was evident that the source of the air leak into the pleural space was from many holes in the visceral pleura and these were seen to be concentrated around the areas of puckering of the lung parenchyma by the tumor involving the visceral pleura The commonest causes of nontraumatic chylothorax are (1) metastatic carcinoma involving the thoracic duct, (2) tuberculosis, (3) unexplained or miscellaneous causes, such as erosion of the duct by aortic aneurysm, occlusion of the duct by blood clot, lymphangiectasis of the duct, and thrombosis of the great vein of the neck.

SUMMARY

1. A case of spontaneous bilateral chylopneumothorax is reported. This was due to a diffuse pleural mesothelioma.

2. A review of the literature revealed no other similar case of spontaneous bilateral chylopneumothorax.

3. The literature revealed no other case of chylous fluid associated with a pleural mesothelioma.

4. This patient did not benefit from attempted decortication of the right lung and ligation of the thoracic duct.

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TRACHEOBRONCHIAL RESTS IN THE ESOPHAGUS

Their Relation to Some Benign Strictures and Certain Types of Cancer of the Esophagus

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THE occurrence of structures lined by tracheobronchial epithelium characterizes a group of congenital anomalies of the esophagus and sets them apart from other known anomalies of this organ. One may list the following as belonging to anomalies with tracheobronchial remnants[1,2]:

1. Tracheoesophageal fistula. This is the commonest anomaly in this group, and the best known.
2. Cysts lined by bronchial epithelium. These may be either mediastinal,[3] or located in the wall of the esophagus.[4,5]
3. Diverticula of the esophagus consisting of tracheobronchial tissue. When such a diverticulum projects beyond the esophageal wall it has been called a lung bud of esophageal origin.[6] In one reported case, the diverticulum was intramural and was termed an abortive tracheoesophageal fistula.[7]

It is apparent that all these anomalies can be regarded as the result of failure of the normal embryonal process of separation of the respiratory tract from the primitive esophagus. In the case of cysts, one might postulate that a tracheobronchial remnant, unconnected to either respiratory or intestinal tract, becomes distended by secretions. The diverticula represent remnants of tracheobronchial tissue with luminal continuity with the esophagus. Despite the simplicity and apparent adequacy of this explanation, other theories of formation of esophageal cysts and diverticula with tracheobronchial epithelium have been advanced and cannot be refuted.[8] These other explanations take two main forms. One is based on the fact that diverticula are normal in certain stages of embryonal development of the esophagus, and that such diverticula might become either pinched off or persist. Furthermore, the embryonic esophagus is lined by cylindrical epithelium, which might persist as such in the anomaly. The other explanation is that the esophageal epithelium is an irregularly vacuolated mass in the 8 week embryo and that anomalies may occur due to the failure of some of these vacuoles to coalesce with the normal esophageal lumen. Here, too, it would be postulated that these abnormal structures retain the embryonal type of epithelium. While at present none of these three theories can be either proved or disproved, it seems to us that the theory of failure of normal separation of the respiratory from the intestinal tract best accounts for the occurrence of these anomalies, and

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especially for the occurrence of diverticula, such as the so-called lung buds and abortive tracheoesophageal fistulas, which consist not only of respiratory epithelium but also of cartilage, smooth muscle, and lymphoid tissue.

While the important role of congenital anomalies in the causation of clinical esophageal disease in infancy is universally recognized, the relationship of embryonal tracheobronchial rests to diseases of the adult esophagus is less well known. In the case of intramural diverticulum referred to above,[7] the congenital rest has been held directly responsible for the occurrence of a localized esophageal stricture. We would like to cite a second such case which was treated by local excision and end-to-end esophageal anastomosis.

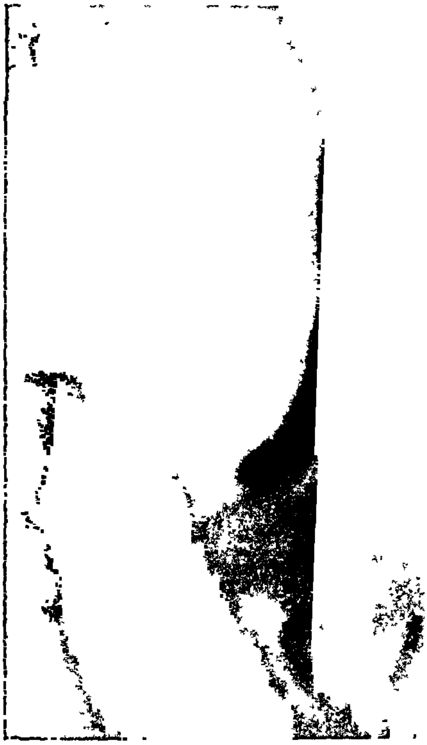


Fig. 1.

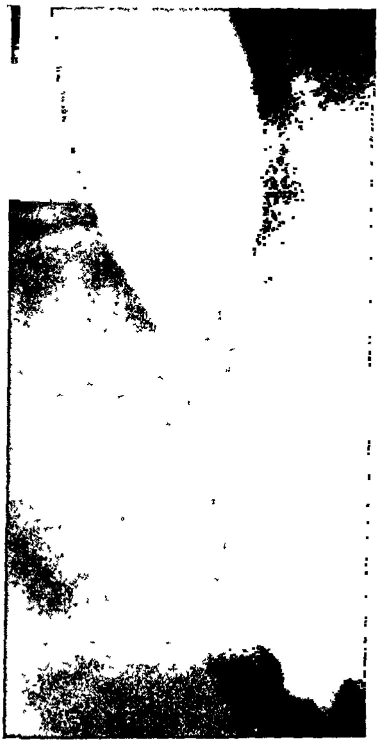


Fig. 2.

Fig. 1.—Case 1. Esophagogram showing negative filling defect due to bolus of food impacted against esophageal stricture.

Fig. 2.—Case 1. Esophagogram showing stricture after disappearance of impacted bolus of food.

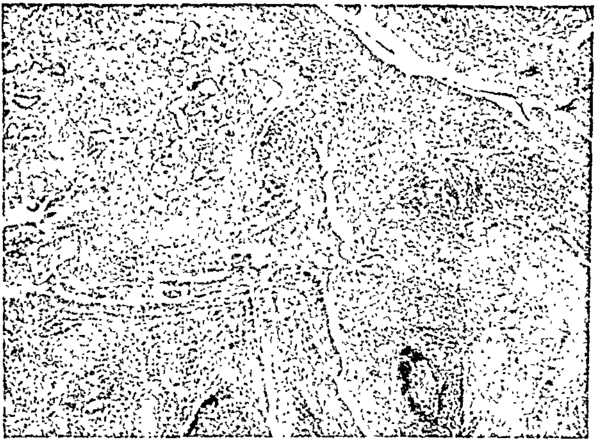
CASE REPORTS

Case 1.—Hosp. No. 232104. S. F., a 57-year-old woman, was first admitted to the Jewish Hospital of St. Louis on April 15, 1953, with the chief complaint of dysphagia since childhood. She stated that in childhood she had had several episodes of complete inability to swallow foods and had had repeated endoscopic manipulations, with successful relief of dysphagia. Some dysphagia had persisted until ten years prior to admission, but during the decade before admission she had been quite asymptomatic. One week before admission, following a breakfast of bacon, eggs and toast, she developed complete dysphagia, with the sensation of food sticking at the level of the lower end of the sternum. The past history was entirely noncontributory.

Physical examination showed a well-nourished woman, who was in no distress. Blood pressure 140/90 mm. Hg, pulse 76, respirations 20, temperature 37° C. The only abnormal findings were the presence of a moderately firm lymph node, measuring about 1.5 cm. in diameter, in the right jugular chain. This was said to have been present unchanged for many years. The liver edge was palpable 2 cm. below the right costal margin and was smooth.

Roentgenograms of the barium-filled esophagus showed a tapering, smooth stricture of the lower third of the esophagus and a negative filling defect thought to represent a bolus of food (Fig. 1). At esophagoscopy a smooth, tapering stricture was encountered at

Fig. 3.



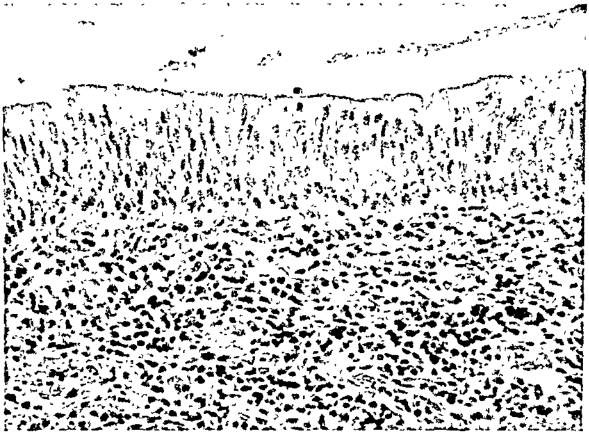


Fig. 4.

Fig. 3.—Case 1. Photomicrograph showing glandlike congenital rests deep in esophageal wall. Note associated lymphoid tissue and mucous glands (hematoxylin and eosin stain, ×35).

Fig. 4.—Case 1. Photomicrograph demonstrating cylindrical epithelium lining the congenital remnant (hematoxylin and eosin stain, ×250).

a distance of 42 cm. from the upper incisors. It did not permit passage of a No. 18 Fr. bougie. A second esophagogram on April 24, 1953, demonstrated that the bolus of food had disappeared and the stricture was now seen to be only about 1 to 1.5 cm. in length (Fig. 2). The patient was discharged on April 25, 1953, to return to the hospital at a later date for definitive therapy.

The patient was re-admitted to the hospital on May 19, 1953. Since discharge she had remained unable to take any but liquid foods. Physical examination was unchanged from the first admission and urinalysis, blood counts, and serologic tests were again within normal limits. On May 21, 1953, a left thoracotomy was done and the short esophageal stricture was found about 10 cm. above the diaphragmatic hiatus. A 3 cm. segment of the esophagus bearing the stricture was excised and esophageal continuity restored by end-to-end anastomosis. The patient's postoperative course was uneventful except for the development of some auricular premature contractions on the ninth postoperative day, which subsided after mild sedation. Esophagogram on the twelfth postoperative day showed a satisfactorily patent anastomosis and, at the time of discharge, on the fourteenth postoperative day, the patient was eating a soft diet.

Gross Description of Specimen.—Pathology No. 54126.* The specimen consists of a previously opened segment of esophagus measuring 2.5 cm. in length. The esophageal wall is firm and measures about 1.0 cm. in thickness. The esophageal mucosa is smooth except in the center of the specimen, where a distinct scar can be seen.

Microscopic Description.—The squamous epithelium is generally intact and in some areas appears thickened. The muscular layer of the esophagus is thickened due to extensive fibrosis. In one section there are two branching structures which have the appearance of very large glands or ducts (Fig. 3). However, they are located deep in the muscularis of the esophagus and they are lined by typical respiratory epithelium, such as is usually encountered in the trachea or major bronchi (Fig. 4). The lumen of these structures is not distended, but contains a small amount of mucus and debris. This would indicate a communication with the esophageal lumen, although this could not actually be demonstrated. The epithelium-lined structures were surrounded by lymphoid follicles and a marked chronic inflammatory reaction. Numerous mixed mucous and serous glands were also associated with these structures and emptied into them.

Comment.—While there is thus no question that benign obstructive lesions of the esophagus can be caused by changes initiated by the persistence of tracheobronchial rests in the esophageal wall, the concept that such rests might give rise to tumors of the esophagus has been advanced in the literature only once.[9] We believe the following case to be an example of a tumor originating in a tracheobronchial rest in the esophageal wall.

CASE 2.—Hosp. No. 560817. H. S., a 65-year-old man, was admitted to the Jewish Hospital of St. Louis on Feb. 1, 1956, because of dysphasia. He stated that he had had indigestion most of his adult life. This had consisted mostly of eructation, gas, and epigastric discomfort. However, previous office records of his referring physician indicated that he had also complained of some dysphagia for many years. He had had repeated roentgenographic studies of the upper intestinal tract, without abnormal findings. About two months prior to admission, he developed marked and definite dysphagia for solid foods, particularly meats, and had to begin washing foods down with water or other liquids. He localized the site of obstruction to the lower part of the sternum, where he also noted some discomfort with radiation to the left shoulder and scapula. The patient denied any weight loss. The past history was entirely noncontributory.

Physical examination at the time of admission showed the patient to be well nourished and well preserved for his age. The blood pressure was 170/80 mm. Hg. Temperature, pulse, and respirations were normal. Abnormal physical findings were limited to the following: the heart extended to the midclavicular line in the fifth intercostal space. There was a soft apical systolic murmur and the second aortic sound was accentuated. Rhythm and rate were normal. The liver edge was palpable 3 cm. below the right costal margin on deep inspiration. It was smooth and nontender. There was a left indirect inguinal hernia.

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*We wish to thank Dr. Herman T. Blumenthal for his help with the pathological examination of this and the following case.

Laboratory findings, including urinalysis, blood counts, fasting blood sugar, and blood urea nitrogen determinations, were all within normal limits. Roentgenography of the barium-filled esophagus showed an obstructing lesion of the esophagus at the junction of the middle and lower thirds. The lesion had the usual characteristics of a carcinoma (Fig. 5). Esophagoscopy was carried out on Feb. 3, 1956, and a fungating lesion, which virtually completely encircled the esophagus, was encountered at a distance of 30 cm. from the maxilla. A specimen taken for biopsy examination was thought to show epidermoid carcinoma.

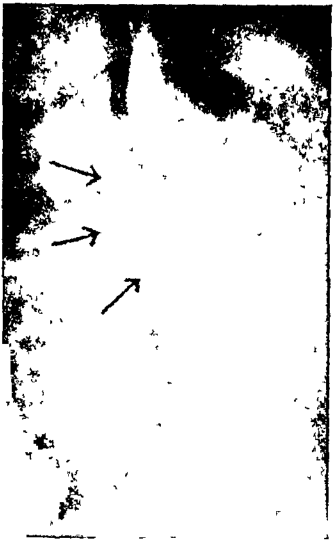

Fig. 5.—Case 2. Esophagogram Arrows outline barely visible soft-tissue shadow caused by the tumor mass.

On Feb. 7, 1956, subtotal esophagectomy, using separate left upper abdominal and right posterolateral thoracotomy incisions, was carried out. The tumor occupied the middle third of the esophagus and esophageal continuity was restored by supra-aortic esophagogastrostomy. The patient's postoperative course was complicated by the development of auricular fibrillation on the day following operation. This responded promptly to digitalization and administration of quinidine. On the sixth postoperative day, the patient was found to be jaundiced. The icteric index was 25 units and bromsulfalein retention was 33 per cent. The jaundice gradually subsided and at the time of discharge on Feb. 23, 1956, the sixteenth postoperative day, the jaundice had cleared and the patient was eating soft foods without difficulty.

Gross Description of Specimen.—Pathology No. 66617. The specimen consists of a previously opened segment of esophagus measuring 13 cm. in length after fixation. About

2 cm. from the superior edge of the specimen there is a tumor with firm raised edges and deep central ulceration. It extends for a distance of 6 cm. in length, leaving about 5 cm. of uninvolved esophagus below the lesion. It virtually completely encircles the esophagus. Eleven lymph nodes were found in the mediastinal tissue attached to the esophagus.

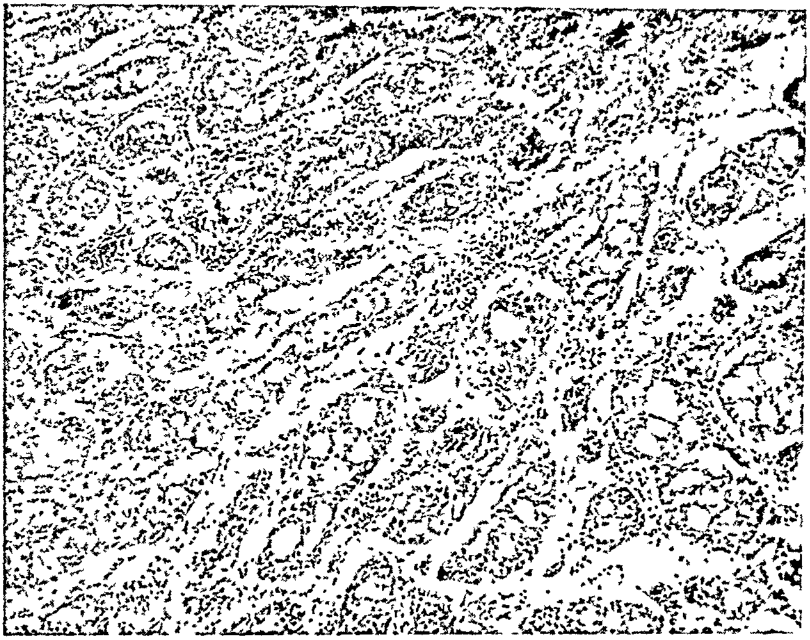

Fig. 6.—Case 2. Photomicrograph of main tumor mass showing characteristic cylindromatous appearance (hematoxylin and eosin stain, ×70).

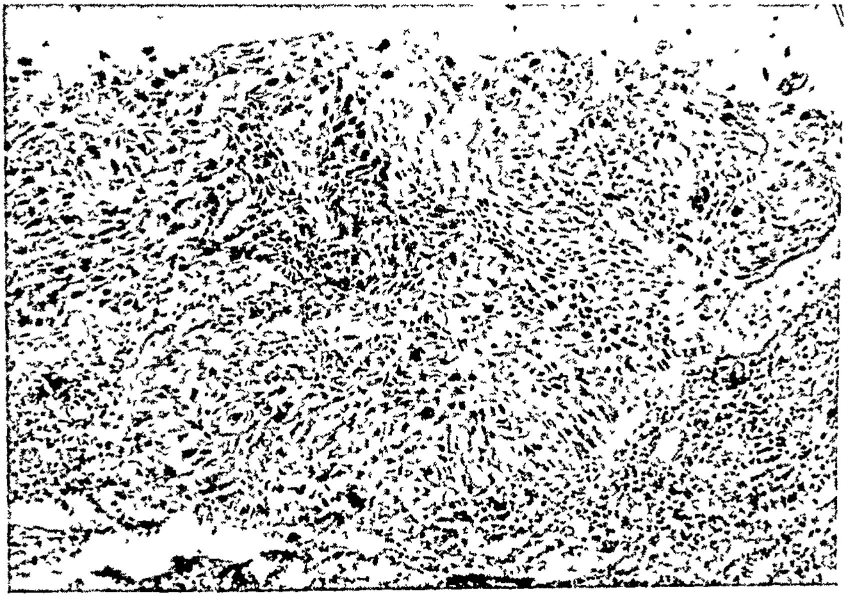

Fig. 7.—Case 2. Photomicrogram of in situ malignant change occurring in squamous epithelium overlying the tumor (hematoxylin and eosin stain, ×175; reduced ⅔).

Microscopic Description.—The tumor is made up of atypical epithelial cells of varying size. The cells have dense hyperchromatic nuclei with prominent nuclear membranes. These cells are arranged in small masses and cords, which often show distinct palisading of cells about a small central mass of basophilic material. The cell masses are separated by a rather loose mature fibrous tissue stroma which has undergone marked basophilic change (Fig 6) In some sections the squamous epithelium over the tumor remains intact and shows in situ malignant changes, consisting of loss of polarity, pleomorphism, and nuclear variation in size and staining quality (Fig 7) However, there are no demonstrable areas of invasive squamous carcinoma, adenocarcinoma, or elements of stromal metaplasia as might be seen in a "mixed tumor" Periodic acid Schiff stains failed to demonstrate any pink staining material Of the eleven lymph nodes, four contained metastatic tumor

The in situ changes of the overlying epithelium were thought to be responsible for the erroneous diagnosis of squamous carcinoma made on the esophagoscopic biopsy specimen

Comment —The main point of interest in this case was the unusual microscopic structure of the tumor It resembled closely the malignant cylindromatous tumors of the tracheobronchial tree[10] It shared with them the finding of areas of more or less intact overlying epithelium, the lack of cornification of tumor cells, the finding of irregular cords and masses of tumor cells in a characteristic stroma, and the distinctive microscopic appearance of the tumor cells A careful search of the literature has failed to turn up any previously published accounts of similar tumors originating in the esophagus The lesion reported as an adenoacanthoma, by McPeak and Arens,[11] deserves special consideration, because in the discussion of this case the authors relate this lesion to salivary gland tumors The apparent similarity to salivary gland tumors rests primarily on the loose stroma which separates noncornifying tumor masses in their specimen However, the authors' descriptions and illustrations demonstrate that the tumor is partly squamous carcinoma and partly glandlike in structure and thus qualifies for the term "adenoacanthoma" In this regard it differs significantly from the tumor reported herein The lack of reported cases indicates the rarity of malignant cylindromas of the esophagus such as our Case 2

DISCUSSION

Speculation as to the origin of this tumor leads us to two hypotheses Since the cylindromatous tumors of the tracheobronchial tree are believed to take their origin in the mucous glands of the tracheobronchial epithelium, one might assume that the tumor described by us arose in the mucous glands of the esophagus In that case one would, however, expect to encounter such tumors with some degree of frequency as is true for the tracheobronchial cylindromatous lesions Therefore we propose instead, an origin in a tracheobronchial remnant in the esophageal wall, such as was found in Case 1 The histogenesis of the tumor in the esophagus would then be identical to its more usual site in the tracheobronchial tree This hypothesis would account for the extreme rarity of the lesion It might also explain the long history of dysphagia with which our second patient presented himself The prolonged dysphagia may have been caused by inflammation around a tracheobronchial remnant in the esophageal wall preceding the development of a carcinoma

If our hypothesis that this carcinoma arose in a tracheobronchial rest in the wall of the esophagus is correct, we might expect to [illegible] the [illegible] ances of tumors characteristic of the tracheobronchial tree occurring in the esophagus. Interestingly enough, 2 cases reported by McKeown[9] fit in exactly with this concept. This author reported 2 cases of typical oat-cell carcinoma, indistinguishable from oat-cell cancer of the lung, occurring in the esophagus. She, too, felt that respiratory tract remnants were the likeliest sources of origin for these tumors. Her cases are also unique in the literature and they strengthen our belief that tracheobronchial remnants in the esophageal wall can cause not only benign strictures of the esophagus, but may also serve as starting points for tumors of the esophagus with the characteristics of primary lung tumors.

SUMMARY AND CONCLUSIONS

There is a group of congenital anomalies of the esophagus characterized by the presence of tracheobronchial rests. These remnants are thought to result from an abnormal separation of the primitive respiratory tract from the esophagus.

A second case of a benign stricture of the esophagus in an adult, due to inflammation around an intramural diverticulum of the esophagus lined by tracheobronchial epithelium, is presented. Treatment was by excision and end-to-end anastomosis.

A further case is reported in which a cylindromatous carcinoma with histologic features quite typical of such tumors in the tracheobronchial tree was found as a primary esophageal tumor. Such a lesion has not previously been reported. However, there have been 2 previous cases of oat-cell cancer identical to those commonly seen in the lung located in the esophagus. We believe that the occurrence of these tumors is best explained by assuming an origin in tracheobronchial rests in the esophagus.

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MORPHINE AND AMIPHENAZOLE FOR POSTOPERATIVE ANALGESIA FOLLOWING MAJOR THORACIC SURGERY

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SATISFACTORY analgesia for patients who have undergone major thoracic surgical procedures is not easy to achieve. The difficulty lies in the fact that morphine and its related compounds, though capable of providing satisfactory analgesia, have shown, owing to their depressant action on the respiratory and cough center, a tendency to produce atelectasis when used alone in sufficient dosage to produce really good analgesia.

The problem of postoperative atelectasis was discussed by Christie, Meakins, and Aronovitch,[1] who studied 23 patients treated with expectorants, postural drainage, bronchodilators, and antibiotics. They felt that pain in the chest on breathing, coughing, and moving was a major factor in the production of atelectasis following operations upon the chest. Bronchoscopic aspiration was necessary in 5 of the 23 patients.

Palmer and Sellick[2] studied a controlled series of 180 consecutive patients who had undergone abdominal surgery. They concluded that a deficient expulsive mechanism, a reduction in bronchial caliber, and qualitative or quantitative changes in the bronchial secretion were all important in the production of atelectasis. They obtained significant improvement in the incidence of postoperative atelectasis by using physiotherapy combined with bronchodilators.

The author of this paper felt that after major operations on the chest, the pain-factor assumed greater importance than in operations elsewhere, and was largely responsible for impaired pulmonary ventilation and ineffective cough. Thus, Björk and Hilty,[3] investigating the arterial oxygen tension of 17 patients after thoracoplasty or pulmonary resection, found an immediate postoperative decrease which they attributed to thoracic pain. This decrease was the greatest and lasted longest after thoracoplasty.

In this Thoracic Surgical Unit, it had been the custom to use morphine very sparingly after pulmonary resections, and not at all after thoracoplasties. In the latter case, pethidine was used in a dosage to be described later. Schaumann[4] described the analgesic effect of 100 mg. of pethidine, compared with 15 mg. of morphine as being about 0.15 1 Patients, particularly after thoracoplasty, often find it extremely difficult to cough because of the associated pain For this reason it was thought that if morphine could be used in effective

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dosage, combined with some agent which would antagonize its respiratory depressant effect, the postoperative course of the patients would be more comfortable and the incidence of atelectasis might be minimized.

Shaw and Shulman,[5] in the treatment of a series of patients with inoperable malignant disease, showed that very large doses of morphine could be given in association with amiphenazole (2:4-diamino-5-phenylthiazole hydrochloride) without the occurrence of undue drowsiness or respiratory depression. They stated that amiphenazole, which was without toxicity, appeared to act as a nonspecific respiratory stimulant, especially when used parenterally. It was therefore decided to use morphine and amiphenazole for the postoperative analgesia of the patients in this investigation.

METHOD OF TRIAL

A consecutive series of 200 *major thoracic operations was studied.* The operations were carried out on 166 patients suffering from active pulmonary tuberculosis. The patients on whom the first 100 operations were carried out (hereafter known as the Control or Control Series) received the standard postoperative treatment, and those concerned in the remaining 100 operations (hereafter known as the Trial or Trial Series) received the treatment under investigation.

Treatment of Control Series.—After lung resections, the patients in this series received 100 mg. of pethidine, if necessary, on return to the recovery ward from the operating theater. On the night of operation, ⅙ or ¼ grain of morphine was given, and thereafter 100 mg. of pethidine every 6 hours for three doses. On the night following operation, the morphine was repeated and, subsequently, three to four more doses of pethidine, 50 to 100 mg. were given if needed.

After thoracoplasty no morphine was given. Pethidine, 50 to 100 mg., was given every 4 hours to a total of 6 doses.

Treatment of Trial Series.—All patients received morphine as often as necessary to relieve pain. Usually this was not found to be necessary more frequently than every 4 hours. The dosage varied from ⅙ to ½ grain and was determined according to the response of the patient. Usually the women patients received ⅙ grain as an initial dose and the men ¼ grain, but as the investigation progressed, the mental make-up of the patients was thought to be more important in determining dosage than considerations of sex or body weight.

The morphine was given intramuscularly and was mixed in the syringe on each occasion with amiphenazole solution. Initially 10 mg. of amiphenazole were used with ⅙ grain of morphine, and 15 mg. with ¼ grain, but later 15 mg. of amiphenazole was a standard dose. Some patients required 30 mg.

A total of 974 doses of morphine with amiphenazole was given to the 80 patients who had 100 operations in this series.

Analysis of Material.—In the Control, the average age of all the patients was 27.7 years, that of the men 33.7 and of the women, 23. In the Trial, the average age of all the patients was 34 6 years that of the men 38 3 and of the women 29 9 There were 86 patients in the Control of whom 38 were men and 48 women, and 80 patients in the Trial of whom 45 were men and 35 women

The average age of the 34 patients who underwent a total of 47 thoracoplasty operations in the Control was 34 5 years (range 18 to 52), and that of the 38 patients who underwent 57 such operations in the Trial 41 3 years (range 19 to 57)

In Table I, "one stage thoracoplasty" refers to a planned one stage procedure in which up to five ribs were removed "First stage thoracoplasty" means an operation in which not more than 3½ ribs were dealt with at one time in a patient from whom it was planned to remove more ribs a fortnight later Two stage operations were, in general, carried out on patients in the older age groups and in those for whom an increased element of risk was thought to be present The discrepancy between the numbers of first and second stage thoracoplasties in the same series is due to the fact that some patients undergoing two stage thoracoplasty have had one operation excluded from the trial due to the consecutive nature of the series, and also because it was not thought advisable to proceed to a second stage in a few patients where this had been originally intended

TABLE I

DESCRIPTION OF OPERATION	NUMBER OF OPERATIONS			
	CONTROL		TRIAL	
	NO	TOTAL	NO	TOTAL
One stage thoracoplasty	15	47	8	57
First stage thoracoplasty	16		24	
Second stage thoracoplasty	14		20	
Resection and thoracoplasty	2		5	
Lobar resection	28	53	14	41
Segmental and wedge resections	14		21	
Pneumonectomy	11		6	
Others*	0		2	2

*The excision of a chronic tuberculous empyema with a chest wall sinus in a man of 60 and a thoracotomy for closure of a postoperative bronchopleural fistula in a man of 25

Palmer and Sellick' (1953), in the paper already referred to, felt that the *quantity of sputum produced was relevant in their cases In this Unit, routine* measurement of sputum-volume is not made There was a predominance of advanced cases in the Trial series which makes it unlikely that there was any significantly smaller volume of sputum produced by the patients in that series In fact, the contrary might be reasonably expected

It is necessary also to consider whether there were any relevant differences in anesthetic technique All patients received a muscle relaxant, intratracheal intubation and controlled respiration in the usual way Premedication was by either morphine and atropine or Omnopon and scopolamine A few patients in the Trial series had premedication with promethazine, chlorpromazine and pethidine, and the author feels that these patients were more difficult to arouse postoperatively and required more amiphenazole than patients otherwise premedicated

These facts indicate that the two series, though differing in the respects referred to, are not so constituted as to bias the results in favor of the treatment under investigation. Indeed the presence in the Trial Series of patients with more extensive disease, of an older age group and with a predominance of thoracoplasties, can, it is thought, be fairly regarded as a bias away from the Trial Series.

Method of Recording Results.—The following classification for the recording of degrees of atelectasis was used:

0—No atelectasis.
1—Slight or moderate atelectasis requiring no special treatment beyond the usual physiotherapy methods.
2—Atelectasis requiring special treatment other than bronchoscopy.
3—Atelectasis requiring bronchoscopy (these patients having had the treatment under 2 without effect).

Price Thomas[6] points out that the incidence of recognized postoperative atelectasis depends a great deal on the frequency with which clinical and radiologic observations are made. The patients in this investigation were x-rayed at least daily during the first two postoperative days, and about twice during the next three to four days. More frequent examinations were carried out as indicated, and the diagnosis of atelectasis was always made initially or confirmed by radiography.

In Grade 2 above, the special treatment referred to consisted originally of the intratracheal instillation of 50,000 units of crystalline penicillin in 2 ml. of sterile water. The injection was made via the cricothyroid membrane as for transcricoid bronchography through an intradermal bleb of local anesthetic. The method produced an explosive cough often successful in bringing about re-expansion of the lung. As this treatment was extremely unpleasant for the patient, it was decided, as the investigation progressed, to determine the effect of the intramuscular injection every 3 hours of 30 mg. of amiphenazole alone for five or six doses. This treatment came to supplant the use of intratracheal penicillin, and was used in the treatment of the last 5 cases in Grade 2 with complete success.

CASE REPORTS

The following two case histories are illustrative of the treatment and results obtained.

Case 1.—A man of 33 years of age had persistent cavitation in the upper lobe of the right lung for which a right upper lobectomy was carried out on May 9, 1956. Postoperatively he had two doses of ⅙ grain of morphine and three of ¼ grain of morphine, each combined with 15 mg. of amiphenazole, between 2:10 P.M. on May 9, 1956, and 9:00 A.M. on May 10, 1956. He was a heavily built man and was found to have a low threshold to pain. Because of this, the dose of morphine was increased to ⅓ grain, with 30 mg. of amiphenazole, at 1:00 P.M. on May 10, 1956. Analgesia was produced for about 3 hours only and, at 5:00 P.M. on May 10, 1956, the dose of morphine was increased to ½ grain with 30 mg. of amiphenazole. This produced satisfactory analgesia for 7½ hours, and, owing to the freedom from pain the patient was able to cough well. Subsequent analgesia was obtained by ⅓ grain of morphine and 15 mg. of amphenazole which was repeated 5 times, and ¼ grain with 15 mg., repeated twice. The postoperative course was entirely uneventful and there was no evidence of atelectasis at any time.

CASE 2.—A man of 46 years of age came to operation on June 11, 1956, for a chronic left sided empyema of about eighteen months' duration which was a sequel to a left spontaneous pneumothorax complicating cavitated pulmonary tuberculosis. At operation the empyema was excised and a resection of the left upper lobe carried out. This was extremely difficult as mobilization of the lung was hindered by the extensive pleurisy. It was anticipated that difficulty might be encountered with this patient's postoperative course. He received 16 doses of ⅙ grain of morphine, 12 of which were given with 10 mg. of amphenazole, and the remainder with 15 mg., from 2:45 P.M. on the day of operation to 11:30 P.M. on June 15, 1956. Breath sounds at the left base were poor, and x ray, on June 15, 1956, showed an opaque left hemithorax due to atelectasis. He was given 30 mg. of amphenazole alone every 3 hours for 5 doses and then every 4 hours for a further 5 doses, intramuscularly. During the next two days the breath sounds returned and serial x-rays demonstrated satisfactory expansion of the residual lower lobe. The subsequent course was uneventful.

RESULTS

The gross results of the investigation are presented in Tables II and III.

TABLE II

GRADE OF ATELECTASIS	NUMBER OF OPERATIONS	
	CONTROL	TRIAL
Grade 0	67	76
Grade 1	18	17
Grade 2	8	7
Grade 3	7	0
Total number of operations	100	100

TABLE III

OPERATION	GRADE OF ATELECTASIS									
	CONTROL					TRIAL				
	0	1	2	3	TOTAL	0	1	2	3	TOTAL
Thoracoplasty, including resection and thoracoplasty	27	11	3	6	47	40	12	5	0	57
Resection, including pneumonectomy	40	7	5	1	53	35	4	2	0	41

DISCUSSION

Table II shows that 7 bronchoscopies were found to be necessary in the Control, and none in the Trial, an incidence of 7 per cent and 0, respectively. The standard error of the two proportions is 2 598 per cent, and the observed incidence of 7 per cent is therefore 2 69 times this error

In Table III, 6 bronchoscopies were found to be necessary among the 47 thoracoplasties in the Control, and none among the 57 in the Trial, an incidence of 12.7 per cent and 0, respectively. The standard error of the two proportions is 4.594 per cent and the observed incidence of 12.7 per cent is 2.78 times this error. In the other cells in Table III there is no significant difference between the Control and the Trial.

Clinically, the treatment made a good impression. The patients were undoubtedly given a much more comfortable postoperative course, particularly after thoracoplasty, and it is noteworthy that 6 of the 7 bronchoscopies in the Control followed thoracoplasty operations. This will be compatible with the experience of most thoracic surgeons. Vomiting, which could be unequivocally ascribed to the morphine, and which led to its discontinuance, was met with in only 2 patients.

SUMMARY

A consecutive series of 200 major thoracic surgical operations for pulmonary tuberculosis has been studied, and a controlled trial of the effectiveness of morphine and amiphenazole, administered simultaneously, in the production of effective postoperative analgesia without danger of atelectasis, has been made.

It is concluded that morphine-amiphenazole therapy is superior to the other routine treatments described, both in the production of satisfactory postoperative analgesia, and in the avoidance of atelectasis of a degree sufficient to require bronchoscopy. In this series the major incidence of postoperative bronchoscopy occurred after operations involving a thoracoplasty, and it is in this group that the treatment described has been found most effective.

It is suggested that morphine with amiphenazole may be of value after extrapulmonary thoracic operations and high abdominal operations, following which atelectasis is regarded as a likely complication.

The use of amiphenazole for the treatment of established atelectasis is described, and it is suggested that this treatment is always worthy of trial before resort is made to bronchoscopic aspiration.

The writer wishes to thank Dr. J. R. Edge for helpful encouragement, and Miss I. Dingwall Fordyce (by courtesy of Professor R. E. Lane) for statistical advice. The operations were carried out by Mr. J. S. Glennie, Mr. P. Jewsbury, Mr. H. A. Daniels, and Mr. N. C. Scott.

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PLEURECTOMY FOR RECURRENT SPONTANEOUS PNEUMOTHORAX

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OBLITERATION of the pleural space is generally recognized as an important feature of the definitive therapy for spontaneous pneumothorax. Obliteration both by closed and open surgical procedures has been incomplete and unsatisfactory in many instances.[1-8] Seeking a solution, we have adopted excision of the parietal pleura to promote fixation of remaining lung tissue to the chest wall. In August, 1951, we treated our first in a series of patients for recurrent spontaneous pneumothorax by thoracotomy, lung repair, and partial parietal pleurectomy. This report includes an additional 13 patients who have undergone fourteen such procedures since that time.

Surgical removal of normal parietal pleura has been proposed for several pathologic conditions. Barrett and Daley,[9] in 1949, suggested bilateral excision of the upper one half of the parietal pleura as an alternate procedure for the technically inoperable patient with tetralogy of Fallot. Blalock[10] agreed that the rationale of increasing the blood supply to the lung by pleurectomy in selected patients had merit. Crenshaw and Rowles[11] have employed parietal pleurectomy in order to revascularize the degenerated, atrophic lungs of patients with pulmonary emphysema. Ellis and Carr,[12] in 1954, suggested pleurectomy be tried on selected patients with spontaneous pneumothorax in whom surgery is indicated. Gaensler[13] recently reported the first series of patients with recurrent spontaneous pneumothorax in whom parietal pleurectomy was performed as an important feature of therapy. Good results were reported in 7 patients, with excellent fusion between the lung and chest wall. He recommended pleurectomy for two groups of patients—those found to have diffuse lung surface involvement, and those with little or no visible gross pathologic lesions at the time of thoracotomy.

In August, 1951, we performed a thoracotomy on a patient with recurrent spontaneous pneumothorax. Lung repair was carried out in the usual manner, by excision and suture of multiple blebs over the apex of the left upper lobe. At this time it occurred to one of us (P.W.G.) that excision of the apical parietal pleura in the region of the disease process would be a feasible method of obliterating the pleural space. This was effected with a good result. In a second patient with bilateral, giant, emphysematous bullae, surgery was indicated for decompression of contiguous good lung tissue. In January, 1952, right thoracotomy, apical and posterior segmental resection, and parietal pleurectomy were done. At a second stage, in July, 1952, left thoracotomy, left

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upper lobectomy, and parietal pleurectomy were performed. Following the second procedure, the patient experienced considerable improvement in respiratory function; over the past four years the patient has continued to be asymptomatic and the lungs are shown to be clear by chest roentgenograms. Consequent to the experience with these 2 patients, excision of the parietal pleura has been instituted as a component part of our surgical treatment for recurrent spontaneous pneumothorax and for giant bullous emphysema. We feel that lung repair with pleurectomy, followed by continuous, active, and complete re-expansion of the lungs is the most reliable method for obliteration of the pleural space. The careful management of the pleural space in the early postoperative period, to ensure complete expansion and fusion of the lung to the chest wall, is as essential as the pleurectomy.

Since January, 1951, 108 patients admitted to this hospital have been treated for 122 episodes of spontaneous pneumothorax. Among these patients, 17 had three or more episodes of spontaneous pneumothorax treated here or elsewhere. Criteria for surgical intervention included recurrence of spontaneous pneumothorax (three or more episodes); demonstration of cystic areas within the lung fields by chest roentgenograms following spontaneous pneumothorax (one or more episodes); or persistent air leak with incomplete lung expansion (ten days or longer) during treatment of the acute episode. Twelve of our patients have been subjected to thoracotomy. Two additional patients operated upon elsewhere by one of us are added to make the total 14.

The indications for surgery are listed in Table I. Ten patients had recurrent spontaneous pneumothorax without roentgenographic evidence of lung field abnormality. In one patient (Case 10) surgery was advised following a single episode of spontaneous pneumothorax associated with cystic areas of the lung field demonstrable on the chest roentgenogram. Another patient (Case 7) was treated surgically after two episodes of spontaneous pneumothorax who also had roentgenographically demonstrable cystic areas of the lung field. Bilateral surgery (Case 11), done in two stages, was indicated for alternating spontaneous pneumothorax associated with bilateral cystic areas of the lung fields demonstrated by chest roentgenograms. In another instance (Case 13), surgery was accomplished during the first acute episode because of a persistent air leak with incomplete lung expansion during intercostal catheter suction therapy. Our total experience with parietal pleurectomy during this 5-year period further includes 6 other patients treated surgically for giant bullous emphysema. Two of these patients underwent bilateral surgical intervention. In all, 20 patients have had twenty-three operations in which parietal pleurectomy was considered an important component of the surgical procedure.

The surgical removal of normal parietal pleura is begun along the cut margins of the pleura, at the edges of the thoracotomy incision. The proper tissue plane is developed by blunt dissection and the pleura stripped in a single large sheet away from the chest wall. The superior dissection is carried over the apex of the chest and down along the mediastinal surface to the azygos vein on

TABLE I CLINICAL DATA

CASE	AGE	SEX	SIDE OF PNEUMOTHORAX			GROSS SURGICAL PATHOLOGY	SURGICAL PROCEDURE	POSTOPERATIVE COMPLICATIONS	PRESENT STATUS
			R	L	TOTAL				
1	26	M	0	3	3	Two blebs apex LUL	Lung repair, apical pleurectomy	None	Unknown
2	20	M	0	3	3	Multiple blebs LUL and basal segments LLL	Lung repair, pleurectomy	None	Unknown
3	21	M	3	0	3	Multiple blebs apex RUL	Lung repair, pleurectomy	Air leak (7 days)	Asymptomatic (24 mo)
4	22	M	0	3	3	Multiple blebs, apex LUL	Lung repair pleurectomy	Rt spontaneous pneumothorax (day of surgery)	Asymptomatic (22 mo)
5	40	F	0	3	3	Necrotic visceral pleura plaque, apex LUL	Lung repair, pleurectomy	None	Unknown
6	41	F	4	0	4	Paper white thin unelastic appearance RUL	Lysis of adhesions, pleurectomy	None	Asymptomatic (18 mo)
7	21	M	2	0	2	Multiple bullae and blebs RUL	Lung repair, pleurectomy	Apical pneumothorax obliterated 4 mo after surgery	Left spontaneous pneumothorax 7 mo after surgery, rt asympt (15 mo)
8	24	M	4	0	4	Single large bullous apex RUL	Lung repair, pleurectomy	Atelectasis, left spontaneous pneumothorax immed postop	Left spontaneous pneumothorax, 3x, rt asymptomatic (14 mo)
9	19	M	3	0	3	Multiple pleural scars apex RUL	Pleurectomy	None	Asymptomatic (13 mo)
10	34	M	1	0	1	Multiple bullae and blebs, apex RUL	Lung repair, pleurectomy	None	Asymptomatic (10 mo)
11	21	M	1	2	3	Multiple bullae and blebs apex LUL Multiple blebs apex RUL	Bilateral surgery, lung repair pleurectomy	Left—none Right—air leak (9 days)	Asymptomatic (6 mo)
12	37	M	1	2	3	Multiple blebs, apex LUL	Lung repair, pleurectomy	Small residual pneumothorax, apex	Rt spontaneous pneumothorax (6 mo)
13	21	M	0	1	1	2 mm peripheral air leak, apex LUL	Lung repair, pleurectomy	None	Asymptomatic (5 mo)
14	30	F	4	0	4	Multiple blebs, apex RUL	Lung repair, pleurectomy	None	Asymptomatic (5 mo)

the right, or aortic arch on the left, where the pleura is excised. A similar dissection of the pleura is carried out inferiorly to the diaphragmatic reflection. No attempt is made to remove the parietal pleura of the diaphragm or pericardium. At the conclusion of the operation, and prior to closure of the incision, large thoracotomy drainage tubes are carefully placed for continuous postoperative suction.

The postoperative complications are shown in Table I. Because of the large raw surface produced by pleurectomy, careful hemostasis must be obtained and postoperative hemorrhage watched for. Blood should be available for administration during and following the operative procedure. Troublesome bleeding has not been a complicating feature in these patients; however, whole blood replacement of an estimated average operative loss of 1,500 to 2,000 c.c. is routine. The complications encountered are not directly attributable to pleurectomy. They have resulted more frequently because of inadequate control of air leaks. The postoperative course was entirely uneventful following nine operations. A somewhat prolonged air leak was present in two instances (Cases 3 and 11) but recovery was uneventful. One patient (Case 4) developed a minimal spontaneous pneumothorax of the unoperated side of the chest the day of surgery; treatment was not necessary and recovery not remarkable. In two instances (Cases 7 and 12), a small, residual postoperative apical pneumothorax was encountered. Of the 15 operations performed only one resulted in a seriously complicated course (Case 8). Immediately following surgery, this patient developed partial atelectasis of the operated lung. Obliteration of the pleural space was therefore incomplete. Bronchoscopy and thoracentesis were required as corrective measures. Further, this patient also had a spontaneous pneumothorax of the unoperated lung shortly after surgery which contributed to his morbidity. One additional, more serious complication worthy of mention was encountered in the group of patients treated for bullous emphysema. Of the 6 patients selected as candidates for surgery, eight operations were done (two bilateral thoracotomies) in which pleurectomy again was an important feature of the surgical procedure. In one of these patients a postoperative bronchopleural fistula developed. This was followed by a *Staphylococcus aureus* empyema. After prolonged hospitalization the patient responded to repeated thoracentesis and Terramycin therapy.

The present status of these patients is likewise listed in Table I. There have been no postoperative recurrences of spontaneous pneumothorax. Eight patients are asymptomatic. One of these (Case 11) required bilateral surgery. In three instances (Cases 7, 8, 12), the postoperative lung is asymptomatic and stable by chest roentgenogram; however, one or more episodes of spontaneous pneumothorax of the unoperated lung have occurred. These patients may require a second-stage surgical procedure on the remaining unstable lung at some future date for complete relief from this condition. Because of a shifting military population, 3 patients (Cases 1, 2, 5) have been lost to follow-up evaluation. However, they were asymptomatic during a 6- to 12-month period of observation.

DISCUSSION

There is a general agreement on the indications for surgery in patients with spontaneous pneumothorax. The most common is recurrent spontaneous pneumothorax, because of the morbiditiy and associated economic loss to the patient. We have limited our selection of patients primarily to those having had three or more episodes of proved spontaneous pneumothorax; 10 of the tabulated patients are in this category. MacQuigg[7] makes a strong plea for earlier surgery for these patients on the basis that gross pathology is usually present and the recurrence rate is high. The second most frequent indication for operative intervention in this experience was one or more episodes of spontaneous pneumothorax with associated cystic areas demonstrated by chest roentgenograms. Three of our patients are in this category. This was the indication for bilateral surgical intervention in one instance (Case 11). Finally, in one patient (Case 13), thoracotomy was necessary because of an uncontrolled air leak during treatment of the first episode of spontaneous pneumothorax. Hemopneumothorax of the spontaneous variety is also considered by some an indication for thoracotomy. The mortality from spontaneous pneumothorax complicated by hemothorax has been estimated to be about 20 per cent.[14] However, we have treated 3 patients with spontaneous hemopneumothorax of moderate degree (500 to 1,000 c.c. of blood removed by intercostal catheter) without resorting to thoracotomy. Possibly the hemothorax produced pleural symphysis; at least there have been no recurrences in these 3 patients. Although we have had no experience in chronic pneumothorax complicated by a fibroplastic membrane which encases the lung, we feel that this condition is also an indication for surgery. This latter condition should occur less and less frequently as more and more physicians become aware of the merits, for the acute episode, of active and immediate re-expansion of the lung by intercostal catheter, water-seal suction drainage of the pleural space.

The predominant gross surgical pathologic lesion found in our patients was the presence of multiple blebs and bullae localized to the apical lung surface. Such was the case at ten operations. In one instance (Case 2), a more generalized distribution of multiple blebs was found. The lung surface of one patient (Case 5) appeared grossly normal with the exception of a single, small area of necrotic visceral pleura at the apex. In another patient (Case 9), the lung surface again appeared grossly normal except for several, small, healed, pleural scars without associated surface blebs. In still another instance (Case 6), the entire upper lobe surface appeared paper-white, thin, and unelastic, witih numerous adhesions indicative of fibrotic change. Finally, in one patient (Case 13) there was a single, 2 mm. air leak in the apex with rolled in fibrotic edges. These characteristic findings of blebs, bullae, and parchment-like changes are similar to some of the gross changes of pulmonary emphysema. They are suggestive of peripheral pulmonary ischemia. Whether this represents a primary or a secondary vascular insufficiency remains to be determined.

One of the fundamental objectives of definitive surgical treatment for recurrent spontaneous pneumothorax is the elimination of the pleural space, which we feel is best accomplished by excision of the parietal pleura. The increased vascularity of the peripheral lung produced by this procedure may be of considerable future benefit to the patient. The incidence of spontaneous pneumothorax in young people appears to be on the increase. One of us (P.W.G.) feels that this probably represents a precursor to the pulmonary changes recognized later in life as emphysema. Spontaneous pneumothorax may be an early clinical manifestation of an atrophic, degenerative alteration of the lung parenchyma. In the beginning this may be localized to an area of involvement, but ultimately destined to progress to more extensive or diffuse pulmonary disease. If they are the result of diminished pulmonary vascularity, progression of these fundamental pathologic changes might be prevented by early parietal pleurectomy. This operative procedure seems to provide an increment of blood supply to the lungs. Therefore, parietal pleurectomy is indicated for the patient with localized pathology as well as the more obvious instance of the patient with diffuse lung surface involvement, or the more rare patient without gross disease demonstrable at surgery. This concept will probably have to remain a theoretical one for years until such time as long-term, follow-up evaluation of patients is possible.

SUMMARY

We have presented a series of 14 patients in whom partial, parietal pleurectomy has been an important feature in the definitive surgical treatment for recurrent spontaneous pneumothorax. The surgical removal of the parietal pleura over the chest wall and superior mediastinum, except for the diaphragmatic and pericardial reflections, as described, is usually not difficult. A pleurectomy limited to excision of the apical parietal pleura, as was carried out in the first patient, we now consider inadequate. The more complete operation was done fourteen times in 13 patients. Pleurectomy is designed to produce two significant changes for the patient: (1) to obliterate the pleural space and prevent recurrence, and (2) to provide revascularization of the peripheral portion of the lung where pathologic changes are usually most marked.

In the majority of patients subjected to thoracotomy for recurrent spontaneous pneumothorax, gross pulmonary pathogenicity, in the form of blebs or bullae, is demonstrated either in localized areas or generalized over the lung surface. If this is a precursor of later pulmonary emphysema, with primary or secondary vascular changes, pleurectomy should be considered more frequently than it has been in the past.

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THE SURGICAL MANAGEMENT OF CONGENITAL BIFID STERNUM WITH PARTIAL ECTOPIA CORDIS

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CONGENITAL bifid sternum associated with partial ectopia cordis is an infrequently encountered anomaly. However, when present this malformation may present a difficult problem in reconstruction. Recently, attention has been directed toward the surgical management of this condition and several procedures have been advocated. In 1947, Burton[1] reported his experience with 2 cases in which grafts of costal cartilage were used to close the defect. Later Maier and Bortone[2] recommended direct surgical closure of the munication is to report an operative technique employed to repair a large defect in infancy and reported a successful case. Two other instances of operation have been reported subsequently.[3, 4] The purpose of the present com- sternal defect associated with partial ectopia cordis in a child, 2 years of age.

CASE REPORT

T. V., a 28-month-old boy, was admitted to the Johns Hopkins Hospital on Sept. 25, 1956, with the history of a bulge in the sternal area since birth. The family history was negative for congenital defects and the child was the result of a normal, full-term pregnancy. The early development was unremarkable except for moderate difficulty in swallowing during the first month of life. The child had been examined first at the age of nine months. At that time he appeared well developed and was in no distress. A triangular defect was noted in the sternal area and the two sides of the chest were connected by a small bridge, one centimeter in length at the xiphoid process (Fig. 1, *A*). The distance between the clavicles was 6 cm. The pulsations of the aorta were visible beneath the skin overlying the defect and, with crying, a marked herniation of mediastinal contents occurred. The remainder of the physical examination was within normal limits. The laboratory findings were not remarkable. At that time it was elected to defer operation until a later date.

On the second hospital admission the history revealed that the child had been asymptomatic and had developed normally during the intervening period. The previously described sternal defect was slightly larger than at the first examination. The child's family was increasingly concerned about the defect and were alarmed at the marked herniation which occurred through it with crying or coughing.

On Oct. 2, 1956, operation was performed. The child was anesthetized with cyclopropane and a midline vertical incision was made from the lower neck to the xiphoid process. Skin flaps were dissected from the underlying pericardium and reflected beyond the sternal bands (Fig. 2). The sternal bands were separated from their posterior attachments to the pleura, and an encircling suture of braided silk was placed temporarily around the sternal bands and approximated. This reduced the size of the defect to approximately

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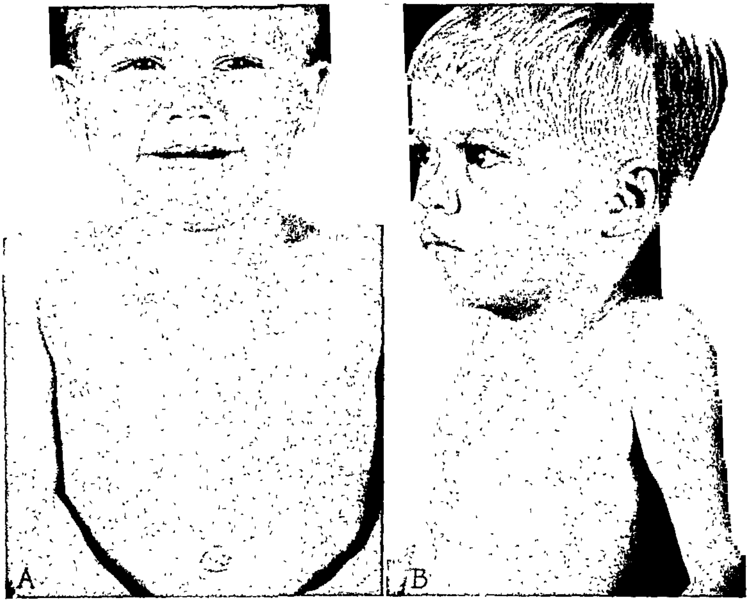

Fig. 1—*A*, Preoperative photograph demonstrating the sternal defect with partial ectopia cordis. With crying, a marked herniation of mediastinal contents is apparent. The two sides of the chest are connected anteriorly by a small bridge at the xiphoid process.

B, Lateral view showing sternal defect during inspiration. The point of union of the two sternal bands is seen at the xiphoid process.

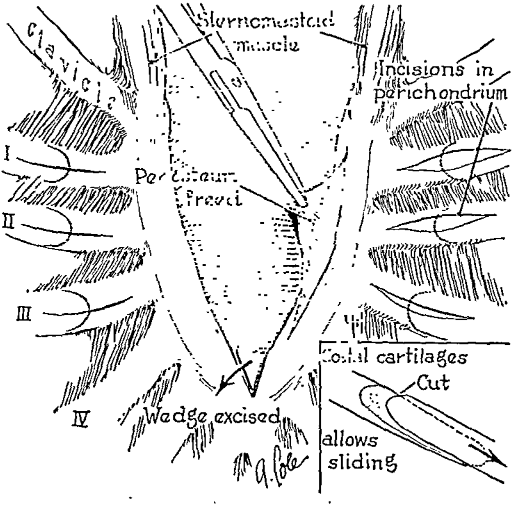

Fig. 2.—Illustration of the defect after dissection of the skin flaps. The sternal bands are shown and the periosteum is being freed from the anterior surface. Incisions have been made into the perichondrium of the first, second, third, and fourth costal cartilages. The inset illustrates the incisions in the costal cartilages.

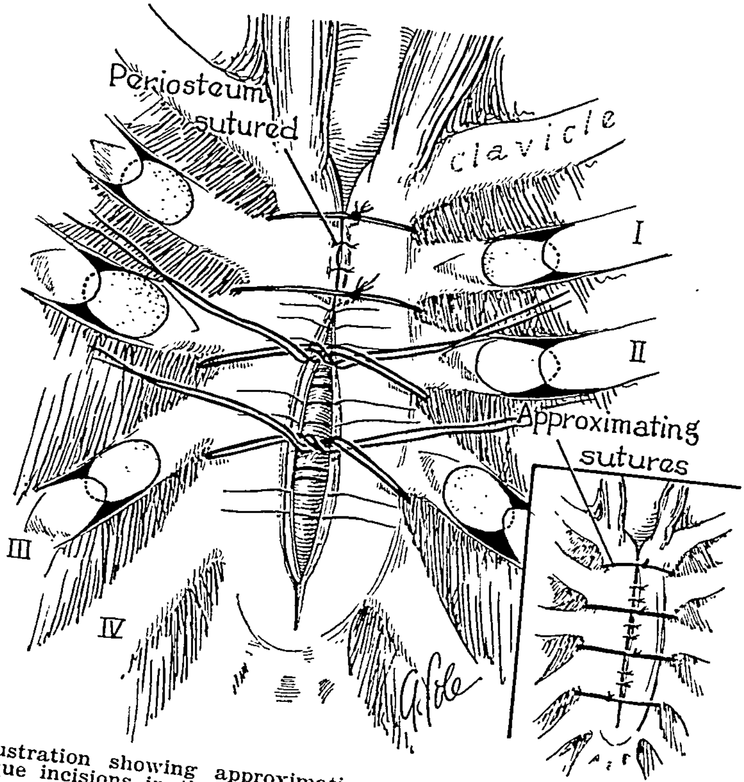

Fig. 3.—Illustration showing approximation of sternal bands with encircling sutures following the oblique incisions in the costal cartilages. Multiple fine silk sutures are shown in the periosteal flaps. The inset shows the final appearance.

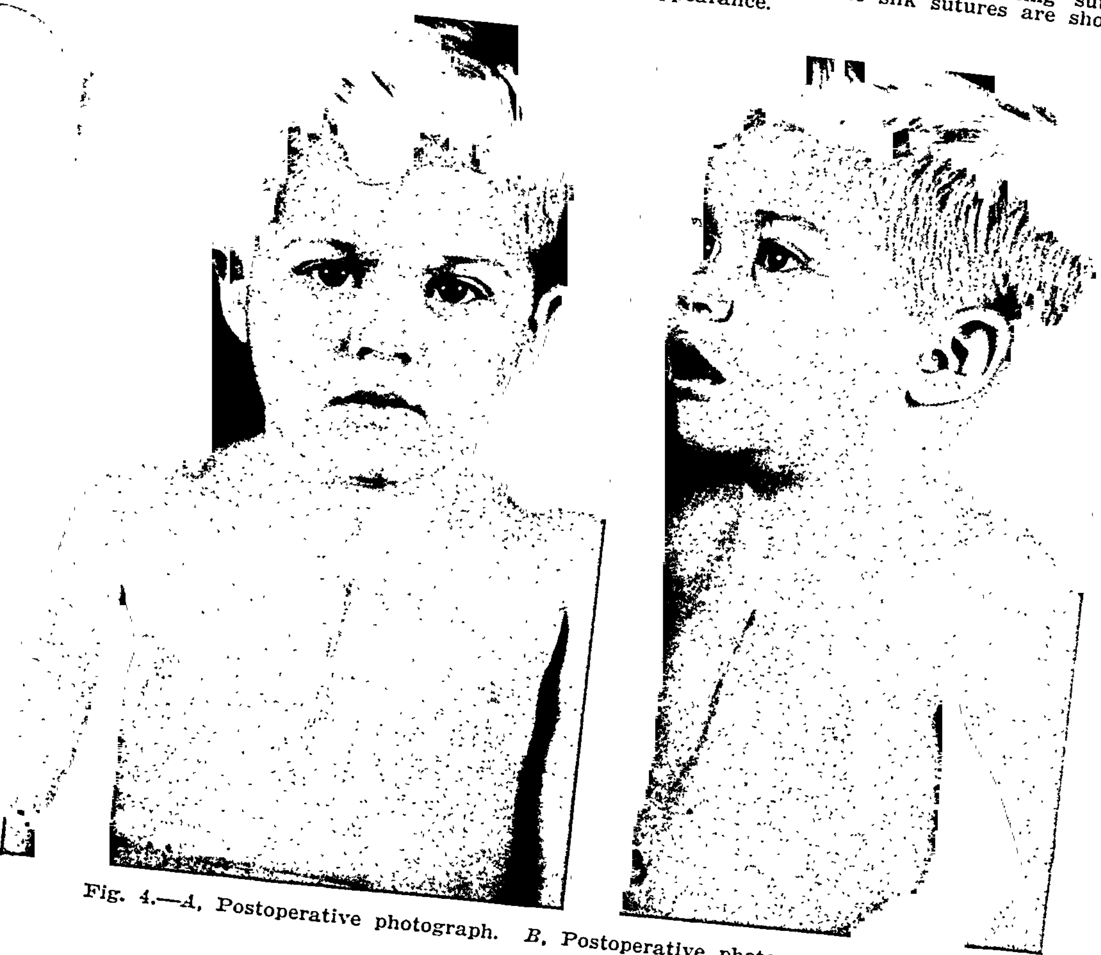

Fig. 4.—*A*, Postoperative photograph. *B*, Postoperative photograph (oblique).

one half of the original dimensions and brought into clear view the attached costal cartilages on either side The costal cartilages of the first, second, and third ribs were exposed and the perichondrium was reflected (Fig 2) An oblique incision was made in the costal cartilages of ribs one, two, and three which allowed sliding, as illustrated in the inset of Fig 2 The periosteum was incised along the medial border of each of the sternal bands and was reflected anteriorly Three sutures of chromic catgut were then placed about both sternal bands and were tied This completely approximated the bands and obliterated the defect The periosteal flaps of the sternal bands were then carefully sutured with fine silk uniting the sternum in the midline (Fig 3) At this point the suprasternal notch was approximately in its normal position After closure of the defect the anesthetist was unable to detect any change in the pulse, blood pressure, or color of the face or upper extremities In addition there was no increased resistance to inflation of the lungs through the endotracheal tube The child made an uneventful postoperative recovery and has since remained well The chest now appears essentially normal and the chest wall is solid (Fig 4, *A* and *B*)

DISCUSSION

Congenital failure of sternal fusion is best explained by a consideration of the embryology of the normal sternum The work of Hanson in tracing the origin and development of the sternum in man and a number of animals has added much to our understanding of congenital bifid sternum He showed that the sternum originates independently of the ribs as two longitudinal bands in the anterior thorax These bands are present in the 6 week embryo At this time there is a space between the two bands, and by the ninth week the two bands have fused in the midline with obliteration of the defect The bands also fuse laterally with the costal cartilages It becomes apparent that in the rare instances of congenital bifid sternum there is a failure of the midline union of these two sternal bands A defect results and through it the pericardium may herniate, being covered only by subcutaneous tissue and skin This condition has been termed either congenital bifid sternum with partial ectopia cordis or failure of sternal fusion with pericardial herniation

In 1947, Burton reported his experience in the surgical treatment of 2 cases of this type His first patient was a child of 7 weeks who had recurrent attacks of cyanosis and dyspnea A block of cartilage was removed from the costal margin and placed over the defect and sutured in place The second defect was in a 12 year old girl with a similar condition The same operative procedure was performed as in the first case and in both the cosmetic result was only moderately good[1] In 1949, Maier recorded the case of a 6 week old infant in which he performed a primary surgical closure[2] In this instance, the two sternal bands were approximated by direct suture and a good result was obtained During the discussion of Maier's paper, Klassen reported a similar case which he had attempted unsuccessfully to repair[3] The most recent report of a surgical repair is that of Longino and Jewett[4] They discuss an infant upon whom they operated at the age of 6 days The defect was closed by primary approximation of the two sternal bands with encircling sutures with a successful result

It appears from the recent literature that the recommended management of congenital bifid sternum with partial ectopia cordis is early operation and primary union of the sternal bands. If operation is performed during infancy, there is no necessity for the use of tissue grafts or plastic materials in the repair of the malformation. In the child reported in this communication the defect was not attacked surgically until the age of two. Nevertheless there were no difficulties encountered at the time of operation and the defect was managed by primary closure. The ease with which this was accomplished is thought to be due to the operative technique employed. A vertical incision was made over the center of the defect and skin flaps were reflected to each side. The sternal bands were freed of their posterior pleural attachments and the costal cartilages which attach to the bands were exposed (see Fig. 2). These costal cartilages were then divided in an oblique manner as illustrated in the inset in Fig. 2. This allowed them to slide and reduce the tension without interfering with the stability of the anterior chest wall. This sliding-type of repair permitted easy approximation of the two sternal bands in the midline by the use of encircling sutures. To strengthen further the midline union, periosteal flaps were reflected anteriorly from each of the sternal bands and were approximated with interrupted sutures. An instance of separation of the sternal bands in the late postoperative period has previously been reported[3] and it is probable that this complication can be prevented by the separate closure of the periosteum.

Despite previous discussions in the literature regarding the difficulty, if not impossibility, in obtaining primary closure of these sternal defects after infancy, the experience reported in the present communication suggests that operation can be simple and effective when it is necessary to perform it at a later date. By use of the sliding costal cartilage technique no difficulty was encountered in the correction of the deformity. In the event a child does not present for surgery until several years of age or in instances in which operation is deferred for other reasons, the operative technique discussed here may be employed with satisfactory primary closure without resort to the use of plastic prostheses or tissue grafts.

SUMMARY

The surgical management of congenital bifid sternum with partial ectopia cordis has been discussed. It is noted that there is general agreement that, when possible, the preferred management is direct operative closure in early infancy. An operative technique is described which permits successful direct closure of the malformation at a later age without the use of grafts or prosthetic materials.

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COARCTATION OF THE AORTA PROXIMAL TO BOTH SUBCLAVIAN ARTERIES

CASE REPORT OF A SIX-YEAR-OLD GIRL

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AS FAR AS we are aware, coarctation of the aorta proximal to both subclavian arteries has not been described previously. One of our patients presented this anomaly, combined with a right aortic arch and a left-sided descending aorta. Preoperatively, no pulse was palpable in either the arms or the legs, whereas the carotids were hyperactive. This case seems to be of interest from the point of view of the surgical technique as well, since it was possible to correct the retroesophageal course of the aorta, so that, postoperatively, it ran in front of the trachea.

Coarctation of the aorta may be present from the middle of the arch to the bifurcation, but in 98 per cent of the cases it is located at the first segment of the descending aorta, immediately distal to the arch.[11] According to Goodson[11] and Mustard and co-workers,[18] coarctation of the aorta is found in one of 1,500 necropsies. A conception of the incidence of this malformation in infants and young children with congenital heart disease is obtained from a survey made by Boeson and Vendel.[10] In 1,137 autopsies on children under 4 years old with congenital heart disease, they found 221 cases of coarctation of the aorta, that is, 20 per cent. Kreutzer[20] made systematic blood pressure measurements in an unselected series of 8,000 children and found 4 cases of coarctation, namely, one in 2,000.

A right aortic arch with a left-sided descending aorta and the left subclavian artery as the last branch has been discussed by several authors as a conceivable anatomic variant.[3, 8, 13, 15] Edwards[8] reported, in 1953, that nine writers had described such cases. Eck,[9] in 1949, stated that a right aortic arch and left descending aorta is a rare variant of right aortic arch; in his series it occurred in only one of 21 cases with a right aortic arch. A right subclavian artery with an anomalous origin, together with a left-sided aortic arch and descending aorta, occurs in 0.5 per cent of all individuals.[16] A mirror image of this condition has been described by Kommerell[17] and by Renander,[19] among others, and in 2 cases by Eck.[9]

The combination of coarctation of the aorta with an anomalous origin of the subclavian artery has been described by Abbott[1] in 2 of the 200 cases of coarctation she had collected from the literature. Another such case was

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reported in 1952 by Brodén, Jönsson, and Karnell[5] among 150 cases of coarctation of the aorta known to them. In these 3 cases, the constriction lay between the left and the right subclavian arteries. Stenosis, proximal to the left subclavian, was described by Abbott in 4 of the aforementioned 200 cases. Edwards,[8] in 1953, gave an account of some similar cases from the literature. He emphasized that they must be distinguished diagnostically from cases with constriction of the first part of the left subclavian and hypoplasia of this artery.

CASE REPORT

The patient was a girl, born on Sept. 5, 1949. Both parents and her older sister were healthy. The mother's pregnancy and parturition were uneventful. Birth weight was 3,450 Gm. Operation for cleft palate was performed in 1952. Immediately after birth she had a few attacks of cyanosis. During her first two years of life she had repeated upper respiratory tract infections but no cyanosis. She was physically underdeveloped; at over two years she weighed only 9.3 Kg. and her height was 79.5 cm. Development was otherwise normal.

She had been hospitalized several times, at which times pulsations were found to be lacking in the radial and femoral arteries on both sides, and a faint systolic murmur was present. A tentative diagnosis of coarctation of the aorta was made.

In June, 1955, on admission to the hospital, the following circulatory signs were found: a network of swollen veins was present on the right side of the neck. The cardiac impulse was heaving and displaced laterally. Faint systolic murmur of stenotic type (Grade 2) was present over the aorta. A sharp first sound was audible over the apex (early systolic click). A stronger systolic murmur (Grade 3-4) occurred over the proximal part of the right carotid. The right carotid was markedly hyperactive, the left normal or somewhat hyperactive. Blood pressure (flush pressure): right and left arm, 85 mm. Hg; right and left leg, 40 mm. Hg.

The electrocardiogram showed left ventricular hypertrophy. Pressure measurement in carotid artery was 140 to 150 mm. Hg. Roentgenologic examination of heart (Drs. Kjellberg and Rudhe) showed enlargement of the left side, particularly of the left ventricle. The heart volume was 10.3 × 6.3 × 6 = 160 c.c., corresponding to 240 c.c. per square meter of body surface.

Angiocardiography (Fig. 1) showed normal anatomy of the right ventricle and of the pulmonary artery and its branches. Both carotids were dilated and were given off from the upper part of the ascending aorta. In the midline, the ascending and descending parts of the aorta formed a sharp angle in an oblique plane; consequently, the probable occurrence of constriction in this segment could not be established definitely in the projections used. The descending aorta was filled almost immediately, indicating the presence of a communication in the stenosis. The superior part of the descending aorta was moderately dilated, as in poststenotic dilatation. An outpouching from the descending aorta probably represented the origin of the left subclavian artery. The internal mammary arteries were not dilated, and collaterals, which were narrow, were present only in the soft tissues of the neck.

Diagnosis: Atypical coarctation of the aorta. The constriction was located centrally in the aortic arch, behind and to the right of the trachea and esophagus, between the origin of the subclavian and carotid arteries.

Operation: Oct. 24, 1955 (Crafoord). When the descending aorta was exposed, the left subclavian artery was found to be given off at the usual site, but to be wide and tortuous. The ligamentum arteriosum ran from the same trunk in an anterior direction. Peripherally to this ligament, the subclavian became narrower. The ligament was divided, and dissection was continued in the mediastinum behind the trachea and esophagus, where the right subclavian

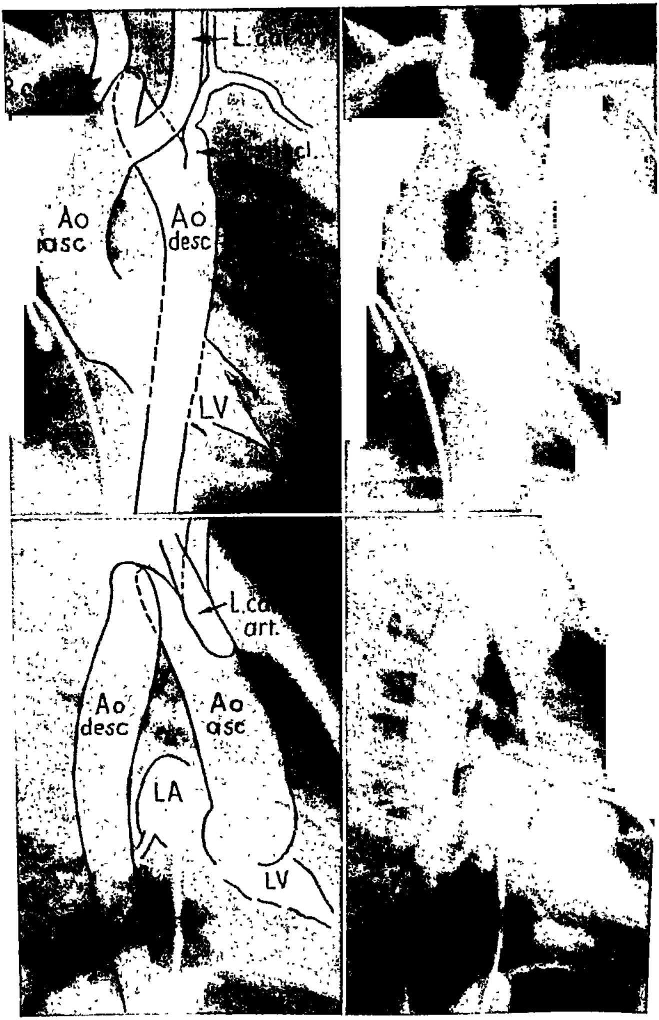

Fig. 1.—Preoperative angiocardiography showing sharp angulation between ascending and descending parts of aorta, dilatation of carotids, both given off from ascending aorta, and diverticulum formation on descending aorta at origin of left subclavian artery.

Ao asc, ascending aorta; *Ao desc*, descending aorta; *L. car. art.* and *R. car art.*, left and right carotid arteries; *L. subcl. art.*, left subclavian artery; *LA*, left atrium *LV*, left ventricle.

was found. The distance between the two subclavian arteries was about 5 cm. The constriction was encountered directly proximal to the right subclavian. After splitting the pericardium, the aortic root was dissected on the anterior aspect; the left carotid artery was first encountered, and then the right carotid, almost at the same level. The coarctation reappeared 3 cm. distal to the right carotid. A Potts clamp was applied to the right subclavian, whereupon a palpable pulse appeared in the right arm, and there was increased pressure in the right subclavian artery, peripheral to the clamp. The right subclavian was ligated and sutured. The aorta was then divided centrally to the coarctation, and sutured end-to-end in front of and lateral to the trachea and esophagus.

The patient withstood the operation well. The pulsations in the right radial artery persisted for the first twenty-four hours after operation, but then disappeared. This must be ascribed to the decreased carotid pressure after correction. On discharge, no pulse was palpable in the right radial artery, but the femoral pulses and left radial pulse were distinctly palpable, although the latter was fainter than normally. Pressure: right arm 85/75 mm. Hg, left arm 100/70 mm. Hg, both legs 100/65 mm. Hg.

A follow-up examination was made in March, 1956. The carotid pulses were softer; the pulse in the left arm and both legs was palpable but faint. Pressure measurement in the carotid artery showed 110 mm. Hg, as compared to 140 to 150 mm. Hg, preoperatively. Angiocardiography disclosed a fairly narrow communication between the ascending and descending aorta, and no postoperative aneurysm.

DISCUSSION

The cardinal symptoms in coarctation of the aorta are: (1) absence of femoral pulsations, and hypertension in the upper part of the body; (2) collateral vessels, evidenced by a systolic murmur on the back, in older children and adults palpable in the interscapular region, and visible roentgenologically as notchings on the lower surface of the ribs; (3) in many cases left ventricular hypertrophy on the electrocardiogram; and (4) in some cases, inward bulging of the aorta at the site of coarctation, seen on the roentgenograms.

In the present case, the diagnosis was based on the absence of pulsations in the arms and legs, and on the high carotid pressure. The anatomic details were demonstrated by selective angiocardiography.

The patient's physical underdevelopment, particularly during her first years, can be ascribed to the deficient blood supply. This also applied to her susceptibility to upper respiratory tract infections. After her second year, her condition improved, and it can be presumed that the collaterals then became better developed. There scarcely seems reason to suspect that she had Turner's syndrome (retarded growth, sexual infantilism, webbing of the neck, and an increased carrying angle of the elbow joints). This syndrome has been discussed in connection with coarctation of the aorta, and is said to increase the risk of operation.[7]

The patient was operated on at 6 years of age. Most authors have hitherto considered that operation should be performed at 10 to 20 years,[10, 14] when sufficient collaterals have developed and before arteriosclerotic changes have appeared in the aorta. Boesen and Vendel's aforementioned autopsy series[10] contained numerous cases of coarctation of both the postductal and preductal type. The authors stated that almost 40 per cent of the patients would have been operable; 50 per cent of these infants died before one year of age. In another series of 90 infants reported from Toronto, 60 per cent of the patients with the postductal type and 89 per cent with the preductal type died before one year of age, most of them even before 6 months.[18] Gammelgaard and Baden[10] therefore concluded that when coarctation of the aorta gives rise to symptoms before 6 months of age, operation should be undertaken immediately.

A tendency to earlier operation can be noted in the literature, the youngest patients operated on being 10 days,[2] 12 days,[18] and 9 months.[10] At our hospital as well, there has been a tendency to operate at an increasingly early age, since it has been found that the aorta and the sutured region grow at the same rate as the individual. For the past seven to eight years, it has been our rule to suggest operation at any time after 3 years of age. In cases with signs of hypertension in infancy, and with electrocardiographic changes, roentgenologic anomalies or clinical symptoms, operation has been advised as soon as the anatomic details have been ascertained. The 2 youngest patients, with coarctation of the aorta operated upon at our hospital, have been 13 and 23 months old, respectively.

Embryology.—The embryologic development of the anomaly in question can be envisaged as follows: In the 2.5 mm. fetus, the dorsal aortae start to fuse upward and downward in the abdominal region, upward to the seventh to fifth intersegmental aortic branches.[6] The ventral aortae become fused to form the truncus arteriosus. Six pairs of branchial arches develop successively between the cranial parts of the ventral and dorsal aortae (Fig. 2, *a*). In the 10 mm. fetus, these branchial arches have already degenerated, with the exception of the third, fourth, and sixth pairs. The third pair becomes the proximal part of the internal carotid artery. The left part of the fourth pair normally forms the aortic arch; its right part becomes partially atrophied and partially included in the proximal part of the right subclavian artery. Its most ventral part becomes the innominate artery. The right and left pulmonary arteries are formed from the sixth pair. Its left dorsal part normally forms the ductus arteriosus (ligamentum arteriosum), whereas its right dorsal part atrophies. The stage shown in Fig. 2, *b* has then been reached. The intersegmental branches from the dorsal (descending) aorta, which form the subclavian arteries, are given off at the level of fusion of the dorsal aortae, but they later shift cranially, so that they generally originate proximal to the ductus arteriosus.

Stenosis and atrophy may occur in other parts of the bronchial arches and aortae. If, with a left aortic arch, this occurs between the right subclavian artery and the right common carotid, it results in a right subclavian with an anomalous origin, given off as the last branch and behind the esophagus. With corresponding atresia, when the right fourth branchial arch instead persists as the aorta, the situation is that shown in Fig. 2, *c*. The left sixth branchial arch then persists as the ductus arteriosus. If, in such a case, the right descending aorta is drawn over to the left and there is, in addition, coarctation between the origin of the right carotid and right subclavian, the conditions are those seen in Fig. 2, *d*, and which correspond to the vascular anomalies in our patient. The collaterals between the carotid and subclavian regions have also been drawn in this figure. Fig. 3 is a schematic drawing of the topographical anatomy in this case. With respect to the right subclavian, the observations at operation showed that it served as a collateral, and that here the blood flowed in a retrograde direction. As far as the left subclavian is concerned, no data are available regarding the conditions before removal of the coarctation. It seems likely that the blood flowed distally after operation, since pulsations then appeared, and have persisted in the left radial artery.

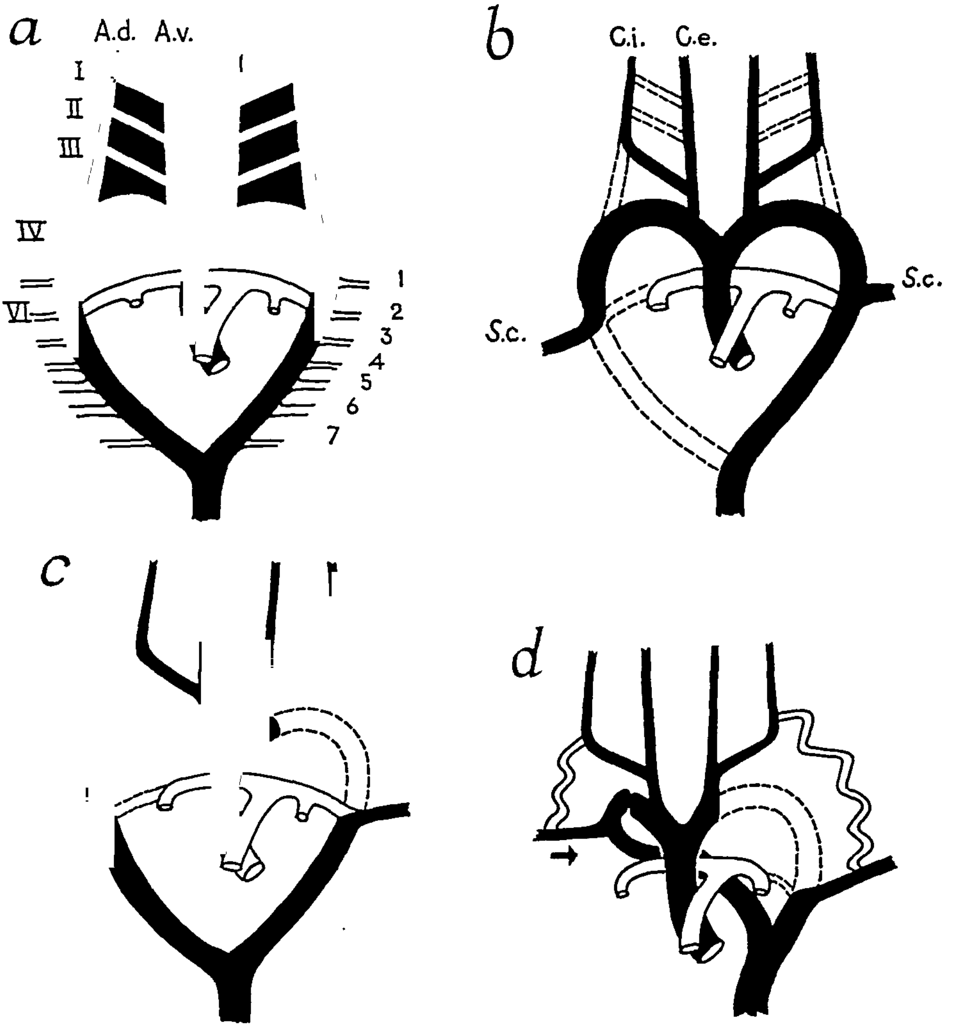

Fig. 2.—*a*, The embryonic stage, when the dorsal aortae fuse to about the seventh intersegmental arteries, and the ventral aortae form the truncus arteriosus.

b, Normal development with left aortic arch and left descending aorta, as well as left ductus arteriosus.

c, Right aortic arch, right descending aorta, left ductus arteriosus, and left subclavian artery with anomalous origin.

d, As in *c*, but with coarctation between right carotid and right subclavian arteries, and dragging of descending aorta to the left. Collaterals between carotid and subclavian regions.

A.d., dorsal aorta, *A.v.* ventral aorta; *C.i.*, internal carotid artery; *C.e.*, external cartoid artery; *S.c.*, subclavian artery; *1, 2, 3, 4, 5, 6, 7*, intersegmental branches of dorsal aorta, of which 5 and 6 become the subclavian artery; *I, II, III, IV, VI*, branchial arches.

According to a theory of Bedford and Parkinson,[4] right aortic arch with left descending aorta is due to one of the three following causes (1) normal development of a left sided ductus arteriosus from the left fourth branchial arch, (2) a left subclavian artery developed from the persisting left dorsal aortic root, or (3) a double aortic arch. In all 3 cases, the right descending aorta is stated to be drawn over to the left, behind the esophagus. No support for the theory of the dragging effect of the left subclavian is found in the material described by Eek.[3] In 2 cases, he found a right aortic arch with a right descending aorta, and the left subclavian behind the esophagus, and between the esophagus and the trachea, respectively.

Our patient had both a left sided ductus arteriosus and a left subclavian artery developed from the persisting left dorsal aortic root. This *may* lend support to Bedford and Parkinson's theory, but does not rule out the existence of some other cause.

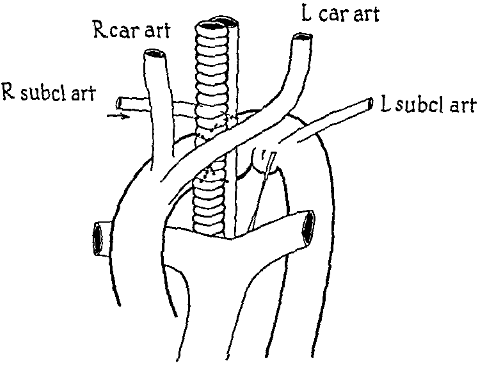

Fig 3—Schematic drawing of the topographical anatomy in the case described. The aorta and right subclavian artery run behind the trachea and esophagus. Both carotids are given off from the ascending aorta. The left subclavian starts with a diverticulum formation from which the ductus arteriosus originates.

R car art right carotid artery *L car art* left carotid artery *R subcl art* right subclavian artery *L subcl art* left subclavian artery

Both the so called adult postductal type of coarctation of the aorta and the infantile preductal type are considered to occur at an early embryonic stage,[7, 8, 13, 16] probably during the second month.[3] Our patient also had cleft palate. This malformation is due to a lack of union between the two palatine processes given off medially from the maxillary processes.[6] In the 25 to 30 mm fetus, the tongue is carried caudally, so that the palatine processes which originally lay beside it come to lie above the tongue. They then grow toward each other and unite in the midline. In the 44 mm fetus, the formation of the bony palate has already started. Thus, both the vascular anomalies and the malformation of the palate fix the time at which the fetal injury [illegible] later part of the second embryonic month.

SUMMARY

1. A case is described of coarctation of the aorta, proximal to both subclavian arteries, together with a right aortic arch and left descending aorta. The diagnosis was made preoperatively by means of selective angiocardiography, among other examinations. A palpable pulse was lacking in both the radial and the femoral arteries.

2. At operation, the coarctation was resected and the aorta, which ran behind the esophagus, was transposed to run in front of the trachea.

3. The patient, who presented a rare variant of coarctation of the aorta, was operated on at 6 years of age. It is pointed out that the recent tendency is to undertake operation of this malformation at an early age. At the authors' hospital, it is done at any time after 3 years of age. When signs of enlargement of the left side of the heart are present, it is performed even earlier, as soon as the anatomy has been determined.

4. The embryology and pathogenesis of the anomalies in question are discussed.

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BUBBLE DIFFUSION OXYGENATOR FOR CARDIOPULMONARY BY-PASS

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RECENT demonstration of the clinical feasibility of extracorporeal circulation for cardiopulmonary by-pass has opened a new era in surgical treatment of cardiovascular disease.[4-6] Development of an ideal oxygenator for the system has been the major variable in the pump-oxygenator systems which have been described since the pumps which are used are relatively uniform in principle. Several methods of oxygenation of blood have been advocated and most have proved to be satisfactory although some have certain practical limitations. Perhaps the simplest method is the bubble diffusion system in which oxygen is diffused through unoxygenated blood. This method provides for adequate mixing of the blood and oxygen with a large surface area of blood being in contact with the bubbles of oxygen. DeWall and Lillehei[3] have described a simple disposable bubble diffusion system made from plastic tubing and consisting of a diffusion cylinder, defoaming chamber, and coiled helix or reservoir. In our experience, which numbers more than 135 cases of cardiopulmonary by-pass, this device was used in 110 and proved to be satisfactory even for flow rates up to 3 L. of blood per minute.[1, 2] Although the DeWall-Lillehei oxygenator is simple and convenient compared to others in use, certain disadvantages exist such as the expense and availability of the plastic tubing, problems of sterilization, and the necessity for assembly before each operation. Thus, having been convinced of the effectiveness of the principle of the DeWall-Lillehei oxygenator, we devised a stainless steel oxygenator based upon the same principle but which was compact, easily sterilized in the pressure autoclave, and readily assembled at operation.

DESCRIPTION OF OXYGENATOR

The oxygenator consists of four principal parts all made from stainless steel (Figs. 1 and 2). The diffusion plate for oxygen dispersal is perforated by 150 drill holes measuring 0.021 inches in diameter and by a large central opening through which the venous blood enters the oxygenating column (*A*). The column is 75 centimeters long and 3.75 cm. in internal diameter (*B*). At the top of the column, blood bubbles out of the overflow into the defoaming chamber

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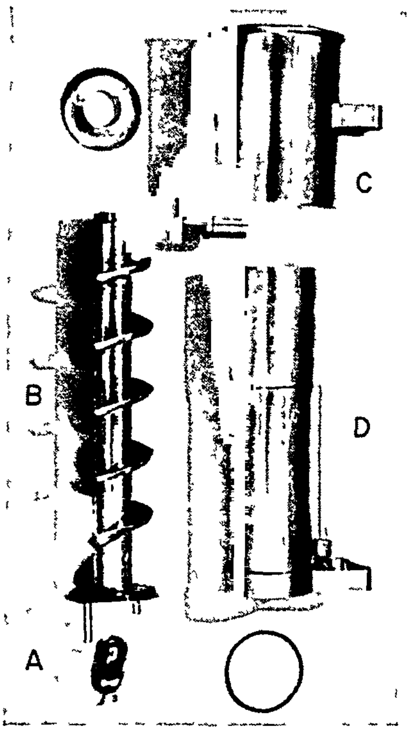

Fig. 1.—Photograph of dismantled oxygenator showing the component parts including the diffusion plate (*A*), oxygenator column with open inclined trough (*B*), defoaming chamber (*C*), and reservoir with level indicator (*D*).

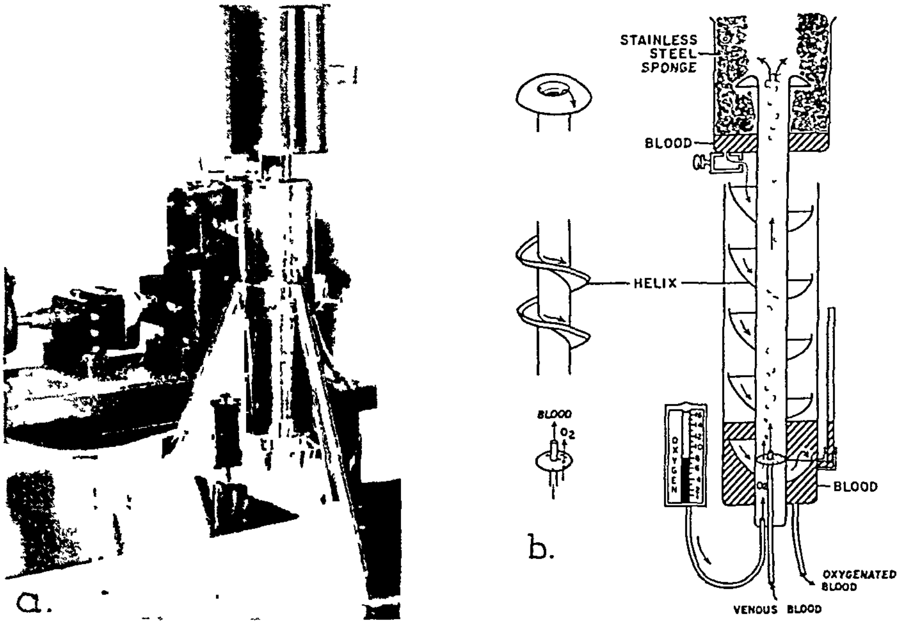

Fig. 2.—*A*, Photograph of assembled oxygenator mounted in tripod holder and connected ready for use. *B*, Cutaway drawing showing method of oxygenation, defoaming, and storage of blood.

(*C*). This chamber measures 15.0 cm. in diameter and contains disposable stainless steel scouring sponges which are coated with Dow Corning Antifoam A spray. Here the blood is converted back to a liquid state and leaves the defoaming chamber through a spigot passing onto an inclined circular trough leading into the collecting chamber (*D*). This reservoir has an attached calibrated stand pipe or level indicator made of glass tubing, the inside of which is siliconized. From the reservoir the oxygenated blood is returned to the patient by way of the outflow pump.

A simple method of determining rate of flow is incorporated into the oxygenator utilizing the level indicator gauge on the side of the reservoir. By closing the valve or spigot under the defoaming chamber for a period of 6 to 30 seconds the volume of blood leaving the reservoir may be measured and the rate of flow per minute calculated. Therefore, it is possible at any time during cardiopulmonary by-pass to calculate the rate of flow.

OPERATION OF OXYGENATOR

The assembled oxygenator is removed from the sterile wrapping and placed on a table so that the apparatus is supported on the handles. The inflow tubes of Tygon tubing for venous blood and oxygen are attached to the diffusion plate assembly under the oxygenator and a tube is attached to the outflow from the reservoir. The oxygenator is lifted into the tripod holder on the pump table and secured firmly (Fig. 2). The antifoam spray is applied to the stainless steel shavings in the defoaming chamber. Warm saline at temperature approximately 112° F. is poured into the chamber and fills the reservoir to warm the entire oxygenator and maintain temperature of the blood during by-pass. Shortly before cardiopulmonary by-pass is commenced, flow rate of the outflow pump is adjusted or calibrated using the warm saline in the reservoir and the level indicator. With the reservoir empty, the inflow pump may be adjusted to the desired flow rate by calculating the rate of filling of the reservoir with blood during the priming process. During cardiopulmonary by-pass, the outflow rate is adjusted to the inflow so that a constant level is maintained in the oxygenator reservoir. Fresh heparinized warm blood which has been maintained at body temperature is used to prime the system and fill the reservoir and oxygenating column. Rate of oxygen flow is set at ten times the calculated flow rate of blood, usually between the extremes of 4 L. and 15 L. per minute.

RESULTS

The oxygenator has been used in 25 patients with congenital cardiac lesions, mostly isolated ventricular and atrial septal defects. Detailed results of these operations will be reported elsewhere, but in no instance has the oxygenator failed to provide for more than 98 per cent oxygen saturation of the blood.[2] Defoaming has also been effective as indicated by samples of blood taken along with the tubing from the outflow connection which have been preserved for more than a week after operation and in which no bubbles have been demonstrated. The largest rate of flow used in these young patients was 1,250 *c.c.* per minute using a rate of 50 c.c. of blood per kilogram of body weight. With slight modifications of the inclined helix, however, rates of flow up to 3 L. or more per minute should be possible. In no instance have postoperative hemorrhagic complications or excessive hemolysis of blood been evident for periods of cardiopulmonary by-pass up to 30 minutes.

SUMMARY

A stainless steel bubble diffusion oxygenator is described which is highly efficient and has the advantages of compactness, convenience, ease of sterilization, and simplicity of assembly at the time of operation. The device incorporates a simple method of determining flow rate through the system.

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FURTHER OBSERVATIONS ON THE LYMPHATIC PICK-UP OF RADIOACTIVE SILVER COATED GOLD COLLOID ADMINISTERED INTRATHORACICALLY TO DOGS

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IN A previous publication,[2] this laboratory reported that radioactive silver coated gold colloid injected into the empty hemithorax of pneumonectomized dogs caused the destruction of the superior mediastinal nodes in 90 per cent of those animals studied No harmful effects from this treatment were noted In this communication, these results will be reviewed along with the results obtained by the mediastinal injection of the colloid at the time of pneumonectomy and following the intrapleural administration of the drug at the time of lobectomy

METHOD

In these experiments, mongrel dogs (weighing 10 to 19 5 Kg) were used They were operated upon under aseptic conditions, using intravenous barbiturate anesthesia At operation or at various times during the postoperative period, radioactive silver coated gold colloid was injected into the pleural space or mediastinum The drug was obtained from the Brookhaven pile and processed after the method of Hahn[1] by the Abbott Laboratories of Chicago, Ill Previous studies[3] showed that it was necessary to use a dose of 40 mc or more in order to produce a consistent radiologic effect on the mediastinal nodes The time interval between operation and injection of the isotope did not alter the results The dogs were sacrificed at intervals ranging from two to forty four days after injection of the colloid material At the time of sacrifice, representative lymph nodes were dissected out, cleansed of adventitial tissue, weighed, placed in standard formaldehyde tubes, and the radioactivity determined by a Texas well counter Histologic studies of the right bronchial, the carinal, the left bronchial, the anterior superior mediastinal, the right superior mediastinal, and the left superior mediastinal lymph nodes were carried out Previous studies[3] revealed that a significant quantity of the colloid material was present in the mediastinal nodes of dogs sacrificed on the second postinjection day, dogs sacrificed later did not have significantly more gold in the nodes studied

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Therefore in calculating the node dosage in beta equivalent roentgens, it was assumed that energy emitted after forty-eight hours could be considered as the lymph node dose.

RESULTS

Of the 10 dogs in Group I that received more than 40 mc. of the drug intrapleurally following pneumonectomy (Table I), it is notable that the superior mediastinal group of nodes received adequate irradiation in 70 per cent of the cases. In 2 dogs, the interval between injection and counting was so long that the counts were not reliable. Histologic study of these nodes, however, showed them to be markedly disorganized. If these nodes are accepted as having received adequate irradiation, the percentage is then 90 per cent. There was one death in this group of animals. Dog No. 18 died on the forty-fourth postoperative day of pneumonia which was thought not to be related to gold therapy. This dog's bone marrow was normal. All animals had well-healed bronchial stumps at the time of autopsy.

TABLE I. GROUP I. DOGS RECEIVING INTRAPLEURAL GOLD COLLOID FOLLOWING PNEUMONECTOMY

NUMBER	DOSAGE (MC.)	β* SUPERIOR ANTERIOR MEDIASTINAL	β SUPERIOR MEDIASTINAL LEFT	β SUPERIOR MEDIASTINAL RIGHT
26	44.4	28,546	5,855	13,270
27	44.4	199,254	181,032	315,675
14	50.0	6,211	96	85
12	50.9	88,632	3,451	2,096
8	52.0	198,077	9,869	48,735
9	52.0	83,220	52,440	96,777
10	52.0	34,940	16,783	Not counted
18	75.0	150,314	Count too near background count	Count too near background count
19	75.0	365,489	Count too near background count	Count too near background count
28	150.0	94,620	86,549	181,716

Taken from Matuska et al.: J. THORACIC SURG. 30: 525, 1956, page 527.

*Radiologic effect is calculated as beta roentgen units.

In the second experiment, 12 dogs were subjected to pneumonectomy; with the chest still open, 45 to 51 mc. (in 10 c.c. of solution) of radioactive silver-coated gold colloid were injected into the mediastinum under direct vision. All

TABLE II. GROUP II. DOGS RECEIVING MEDIASTINAL INFILTRATION OF GOLD COLLOID

NUMBER	DOSAGE (MC.)	β SUPERIOR ANTERIOR MEDIASTINAL	β SUPERIOR MEDIASTINAL LEFT	β SUPERIOR MEDIASTINAL RIGHT
5	50	51,700	42,043	48,154
6	51	31,500	16,700	169,800
7	51	15,200	32,000	76,200
8	51	69,000	10,500	25,100
9	49	13,700	2,000	12,900
10	49	76,800	90,400	81,900
11	49	58,000	146,900	76,100
13	50	50,700	299,500	102,800
15	45	109,700	145,800	24,300
18	50	16,500	79,200	54,300
19	50	55,800	8,200	204,500
22	50	40,100	184,000	114,400

of these animals showed histologic destruction of the superior mediastinal group of nodes. Radioactivity was also high, as determined by the Texas well counter (Table II). However, 3 dogs (25 per cent) showed evidence of a bronchial stump fistula.

In the third experiment, 10 dogs were subjected to lobectomy. At the time of operation 35 to 50 mc. of the isotope were placed in the pleural space. The residual lung was inflated and the chest was closed without drainage. These dogs were sacrificed from seven to ten days following the procedure. Nine of the 10 dogs (90 per cent) had significant irradiation of the superior mediastinal group of nodes as determined by the Texas well counter (Table III). On histologic study, all the superior mediastinal nodes showed varying degrees of radiation effect There was one death in this series. Dog No. 21 died on the fourteenth postoperative day following a left upper lobectomy. At autopsy, pneumonia was present in the left lower lobe and there was atelectasis on the contralateral side. It was felt that this dog died as a result of the procedure, but *not* as the result of the gold therapy. Except in the dog that died, the remaining lobe on the operated side was free of abnormalities at the time of autopsy.

TABLE III. GROUP III. DOGS RECEIVING INTRAPLEURAL GOLD COLLOID AT TIME OF LOBECTOMY

NUMBER	DOSAGE (MC.)	β SUPERIOR ANTERIOR MEDIASTINAL	β SUPERIOR MEDIASTINAL LEFT	β SUPERIOR MEDIASTINAL RIGHT
12	50	55,400	25,500	19,900
14	50	121,500	251,900	Count too near background count
16	45	81,000	93,100	40,800
17	50	Count too near back ground count	Count too near back ground count	6,100
20	50	112,600	110,900	180,100
21	50	194,400	6,300	36,300
23	50	223,800	84,000	256,600
24	50	94,500	26,500	305,200
25	35	177,000	557,000	389,700
26	35	290,000	219,000	384,300

The histologic changes in the mediastinal lymph nodes of all three groups can be considered together. There was total necrosis of the nodes and the immediate surrounding tissues in those instances in which the radiologic effect was the greatest In the less severe cases, the nodal architecture was partially preserved, but with a complete loss of lymphocytes; plasma cells and reticulum cells were prominent survivors in these nodes. Erythrophagocytosis was noted in lymph nodes with high counts in animals that were sacrificed early. It is notable that the vascular damage frequently assumed a form which strikingly resembled fibrinoid necrosis.

DISCUSSION

One can conclude from these experiments that the lymphatics of the parietal and mediastinal pleurae in dogs drain into the mediastinal lymph nodes bilaterally. These nodes may be destroyed by the intrapleural or intramediastinal injection of radioactive silver-coated gold colloid. It is felt that if these observations prove to be valid in the human being, a prophylactic type of mediastinal dissection may be added to pneumonectomy in the treatment of patients with resectable bronchogenic carcinoma. Although we do not think it likely that lymph nodes replaced by tumor would pick up sufficient radioactive material to destroy them, it is not unreasonable to believe that small tumor emboli in these nodes could be destroyed by this method. We do not advocate the mediastinal injection of this drug in human beings because of the high incidence of bronchial stump dehiscence in dogs.

Six *patients have been treated with radioactive silver-coated gold following* pneumonectomy for brochogenic carcinoma at the Thayer Veterans Administration Hospital. No ill effects have been observed from this therapy. Dosage has ranged from 75 to 150 mc. Three of these 6 patients are alive, eight, twenty, and twenty-seven months following therapy, without evidence of recurrent tumor.

SUMMARY

1. Radioactive silver-coated gold colloid consistently produces a radiologic effect upon the mediastinal lymph nodes of dogs following intrapleural injection and following injection into the mediastinum.

2. No harmful effects have been noted following intrapleural administration. The incidence of bronchial stump dehiscence following the intramediastinal injection of the colloid in pneumonectomized dogs was 25 per cent.

3. Currently we are treating human beings with the gold colloid following resective surgery for bronchogenic carcinoma.

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Original Communications

VISUAL REPAIR OF CONGENITAL AORTIC STENOSIS DURING HYPOTHERMIA

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CONGENITAL obstruction to left ventricular outflow, either at the aortic valve or at subvalvular level was formerly considered to be both relatively uncommon and benign Abbott,[1] in reporting 1,000 cases of congenital heart disease, *found 18 instances of subvalvular and 11 of valvular* aortic stenosis Nadas[2] stated that these 2 lesions accounted for slightly over one per cent of his patients with congenital heart disease Campbell and Kauntz,[3] however, reported 40 patients and emphasized that the lesion is more common than is generally thought The 11 surgical patients in our present series were selected from a total of 57 patients with congenital valvular or subvalvular stenosis These 57 patients represent about 5 per cent of the total individuals observed in our clinic with congenital heart disease during the past 6 years Certainly the incidence is far below that of septal defects but, nontheless, obstruction to left ventricular outflow is not a rare lesion

The benignity of this disease has also been subject to some question in recent years In a group of 37 patients, Downing[4] reported three instances of sudden death Nadas[5] encountered 5 such accidents in a group of 67 patients and, more recently, Braverman and Gibson[6] reported comparable figures with 6 cases of sudden death in a series of 73 patients followed over an 8 year period, an incidence of 8 2 per cent We have had 3 cases of sudden death in our group of 57 patients, or 5 2 per cent It is apparent, therefore, that the lesion is far from benign and a safe and effective operative procedure would have much to recommend it

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The first operative attacks on congenital obstruction were transventricular, using instrumentation similar to that employed for rheumatic aortic valvular stenosis. Bailey[6] stated that the small aortic lumen in children makes this approach technically easier than the closed transaortic route, since the vessel is too small to accommodate the finger. Marquis and Logan[7] reported 6 patients operated upon via the ventricle. All but one patient had postoperative aortic insufficiency which was considered to be severe in 2, moderate in 2, and slight in one. Downing[4] reported 19 operative patients with one operative death and one death due to a cerebral vascular accident several months postoperatively.

Experimentally, open operation on the aortic valve, using circulatory occlusion, was first described by Carrel[8] in 1914. Apparently little further study of the problems involved were undertaken until the work of Clowes and Neville[9] in 1954. In accord with our often expressed opinion that most, if not all, intracardiac operations can better and more safely be done under direct vision, an experimental technique for the transaortic exposure of the valve during circulatory arrest and hypothermia was evolved in our laboratory in early 1955. Emphasis was placed on methods to prevent trapping of air in the heart to avoid coronary air embolism. The first patient to have a successful direct vision procedure on the aortic valve was operated upon Nov. 17, 1955. The experimental technique and the course of the patient have been previously reported.[10] Very shortly thereafter, Lewis and his associates,[11] also using circulatory occlusion during hypothermia, successfully operated upon a patient with congenital aortic stenosis by the transarterial open approach. Kay and Morrow[12] employed this method in a patient with subaortic stenosis.

A different method for the open transaortic approach to the aortic valve has also recently been developed and applied clinically. That the heart might be nourished by retrograde venous perfusion was first suggested by Pratt[13] in 1898, and subsequently demonstrated by Roberts, Browne, and Roberts[14] in 1943. Experimentally, Blanco, Adam, and Fernandez[15] developed a method for general body perfusion combined with retrograde perfusion of the coronary sinus which allowed exposure of the aortic valve. Based on these studies, Lillehei and co-workers[16] have applied the method clinically. This technique has the advantage of providing a somewhat longer safe period for the open procedure, but has the disadvantage that the operative field instead of being dry is constantly flooded by coronary blood flow.

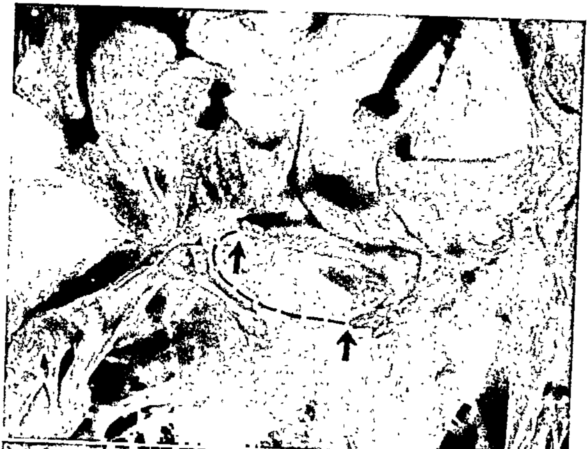
The 11 patients presented here were all operated upon using general hypothermia, circulatory arrest, and open, transaortic plastic revision of their stenotic lesion.

PATHOLOGY

Three of the 11 patients had subvalvular obstruction while 8 had valvular stenosis.

Subvalvular stenosis in all 3 patients consisted of a ring or collar of firm, whitish, fibrous tissue encircling the outflow tract of the left ventricle. This ring was located from 1.0 to 2.0 cm. below the valve in each instance. The ring involved the lowest edge of the right valve cusp in one patient (Fig. 1). The

Fig. 1.



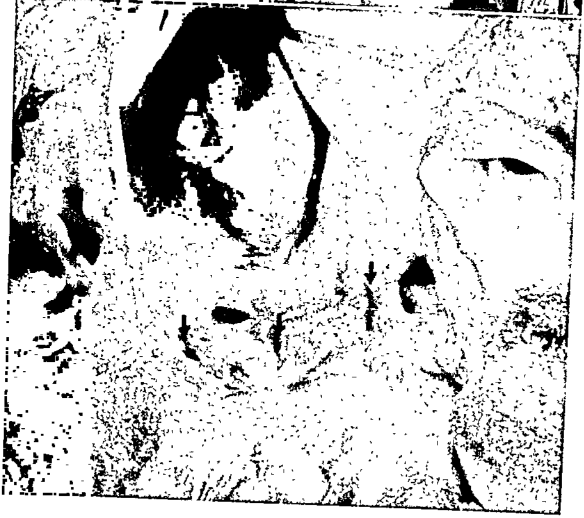


Fig. 2.

Fig. 1.—The autopsy specimen from a patient with subvalvular stenosis who died of tracheobronchitis 5 days after operation. The remaining portion of the fibrous constricting collar is seen to lie just below the cusps of the aortic valve. The dotted line indicates the extent of the resection at the time of surgery.

Fig. 2.—The autopsy specimen from a patient who died on the twenty-seventh day following operation of hemorrhage from the aortic wound. The arrows indicate the sites of incision. The two lateral ones were properly placed in the commissures, but the central one created moderate insufficiency since the commissure (not seen behind the cusp) did not extend completely up to the lip of the cusp.

posteromedical attachment of these obstructing collars was across the ventricular aspect of the aortic leaflet of the mitral valve. This is a relationship of extreme importance as well be discussed later. There was no definable infundibular chamber observable in the left outflow tract distal to the obstruction. Of great importance, however, was the uniform participation of the valve and the valve ring in the phenomenon of post-stenotic dilatation. This dilatation was already established in the intramyocardial aorta so that the diameter of the valve ring was considerably greater than normal. The sinuses of Valsalva were also grossly enlarged and the valve cusps were moderately thickened. This participation of the valve and the most proximal portion of the aorta in the post-stenotic dilatation may contribute a radiographic contour suggesting the subvalvular location of the lesion and also permits operative exposure through the valve ring via the aortic incision. In one patient, a small patent ductus arteriosus was present in association with subvalvular stenosis.

Valvular stenosis was similar, in general, to the Type II stenosis of the pulmonary valve which we have previously described.[17] The cusps were formed, and thickened fused commissures existed. The opening in the valve in all patients was remarkably constant, averaging about 8 mm. in diameter. The commissures were at times irregularly fused so that the valve orifice lay somewhat eccentric. The cusps, and most particularly the free edge of the cusps, were thickened and firm. However, in no patient was calcification present. We did not encounter a valve similar to the Type I pathology in which only rudimentary commissures were present and the valve was an inverted funnel. This, however, has been described.[18]

Bicuspid valves were found in 2 of the 8 patients with valvular stenosis, and another had essentially a biscuspid valve except that the third commissure was partially formed. In the truly bicuspid valve, the orifice was a slit-like structure, and the surgeon had the impression at operation that incision of the commissures had minimal effect upon the capacity of the thickened valve cusps to open more widely. The incompletely formed commissure in the third patient with a bicuspid valve came to within 2 mm. of the free edge of the cusp and thus created the illusion of a tricuspid valve (Fig. 2). Incision of this incompletely formed commissure resulted in significant postoperative regurgitation. It is extremely important, therefore, that the operator realize the possibility of incomplete formation of commissures and avoid incision into such commissures if they are present.

The post-stenotic dilatation beyond valvular stenosis began just distal to the uppermost attachment of the commissures to the wall of the aorta. The vessel was dilated to approximately twice its normal size in the ascending portion and then gradually tapered to normal in the first portion of the arch. This dilatation is not associated with hypertrophy of the aortic wall. Indeed, the vessel in this area is thinner and more friable than in the descending aorta. Thus, the dilatation enhances the ease of application of tangential noncrushing clamps; the narrow segment just above the valve renders exposure of the valve more difficult; and the friable nature of the aortic wall makes closure of the incision both difficult and dangerous.

CLINICAL CONSIDERATIONS

The relative frequency of subvalvular as compared with valvular stenosis has been difficult to ascertain in the past. Certainly the only conclusive answer heretofore depended upon necropsy findings, as even an operative approach through the left ventricle would not decide this question. The clinical literature relative to this subject, therefore, has led to considerable confusion. Thus, some years ago, when it was popular to consider the obstruction as usually subvalvular in origin, reports by Young[23] and Brofman and Feil[22] described the classical picture of aortic obstruction and considered it to be due to subaortic stenosis. It would seem probable, at the present time, that the majority of these patients had the obstruction at the valvular level. More recently, the identical clinical picture has been considered to reflect aortic valvular stenosis.[3, 4, 22]

There has also been considerable confusion as to the etiology of aortic valvular obstruction. Some of the patients presented as having congenital aortic valvular stenosis by Campbell and Kauntz[3] might be considered to be of rheumatic or dengenerative origin by other observers. However, since it is apparently true that a patient with relatively mild aortic valvular stenosis may develop degenerative changes and thus present, in later years, with calcific aortic stenosis, it is difficult if not impossible at this stage to determine the exact etiology from clinical evaluation. It is evident that in order to be certain that the valvular stenosis is congenital in origin, one would have had to evaluate personally the case prior to the tenth year of life and to have found the classical clinical picture of aortic obstruction without any history of rheumatic disease. Even the history of a murmur from childhood in a patient 40 years of age is not adequate background upon which to make a diagnosis of a congenital deformity, as the murmur might have had a different origin.

The patients in the present series were all below 15 years of age, with but one exception, and a murmur associated with the classical clinical picture was noted prior to the age of 12 in all but 2 patients. The actual pathology was also observed in a dry field at the time of surgery in all patients. Therefore, in this present series, no doubt exists as to the congenital etiology of the lesion, nor as to the site of the obstruction.

Symptomatology.—Reliance on symptoms in evaluating the severity of obstruction in children with congenital aortic stenosis is not rewarding and, actually, may be most misleading. Relatively severe obstruction may exist without any symptoms whatsoever. The presence of dizzy spells and bouts of syncope are ominous symptoms and may presage sudden death. On the other hand, sudden death may occur without any previous symptom. The finding of decreased exercise tolerance is at times noted, but this is frequently difficult to assess in a child. The development of congestive failure is uncommon, but does occur. Thus, in general, the problem is similar to evaluating patients with pulmonary valvular stenosis and an intact ventricular septum, in whom symptoms are also misleading. In both instances, the patients may have severe obstruction and yet have minimal symptomatology. In the over-all clinical evaluation of the patient, therefore, the physical findings, electrocardiogram, and fluoroscopic examination are the important factors in the evaluation of the severity of the obstruction.

Physical Findings.—The classical physical findings have been well outlined by previous authors.[2, 3, 22, 23] The body habitus is usually normal. A systolic thrill to the right of the upper sternum and transmitted into the neck vessels is almost always present, as it was in all patients in our group. The thrill may be rather diffuse over the precordium, but is usually maximum to the right of the upper sternum. Palpation of the apical area of the precordium will frequently reveal a forceful sustained thrust that is associated with left ventricular hypertrophy. The peripheral pulse may be entirely normal or may be small, and the blood pressure and pulse pressure may be normal, or there may be a somewhat lowered systolic pressure and a decreased pulse pressure. These findings vary, in general, with the severity of the aortic obstruction.

Auscultation reveals a loud harsh systolic murmur, well heard at the base, and usually of maximum intensity in the first or second right intercostal spaces at the sternal border and well transmitted into the neck vessels. In the patients with subaortic stenosis, we believe that the murmur is frequently of maximum intensity somewhat lower in the third intercostal space and beneath or just to the left of the sternum. This was true in the 3 patients with subaortic stenosis in this series. A diastolic murmur of aortic insufficiency was not present in any patient in this group.

In distinguishing subvalvular from valvular stenosis, the intensity of the second aortic sound has not proved to be of any value. In congenital valvular stenosis, the second aortic sound is usually of entirely normal intensity and may at times be increased. The reasons for the latter finding are certainly not clear.

Electrocardiography.—The electrocardiogram offers valuable information in determining the severity of the obstruction. Evidence of left ventricular hypertrophy is considered to be a most reliable sign of the severity of the aortic obstruction. The electrocardiogram, however, may be within normal limits even in the presence of definite hypertrophy of the left ventricle, since it is not a very sensitive reflection of the degree of left ventricular hypertrophy. Therefore, the definite presence of left ventricular hypertrophy in the electrocardiogram implies significant stenosis, and the earlier in life that this is present, the more significant is the finding. In addition, to the changes in the QRS complexes, reflecting left ventricular hypertrophy, the T waves are usually inverted in the left precordial leads. Thus, the changes are suggestive of the so-called "systolic overload pattern," as described by Cabrera.[24] The electrocardiogram is particularly valuable, therefore, in the patient with no symptoms and a heart that is within normal limits as regards over-all size. Severe stenosis and hypertrophy of the left ventricle may be present, reflected only by the electrocardiogram.

X-ray and Fluoroscopy.—The main pulmonary artery and its branches were considered to be normal in size and amplitude of pulsations in all patients. Dilatation of the ascending aorta has been mentioned by most observers as being a prominent feature of the fluoroscopic evaluation of patients with aortic obstruction.[2, 4, 9, 22] It is considered that the site of the post-stenotic dilatation may be of some aid in differentiating subvalvular from valvular obstruction. At surgery, we have noted that in subvalvular stenosis, the most proximal portion of the aorta including the sinuses of Valsalva take part in the post-stenotic dilatation, whereas, in valvular stenosis, the post-stenotic dilatation is noted to take its origin distal to the sinuses of Valsalva. Therefore, at the time of fluoroscopy, this was looked for very carefully. However, this is very difficult to evaluate with any degree of accuracy, since the very proximal dilatation lies within the shadow of the heart. However, in the 26-year-old patient with subaortic stenosis, the low dilatation was clearly noted on fluoroscopy and appeared as a double contour within the cephalic portion of the right atrial shadow. The patients with valvular aortic stenosis revealed a tendency for the post-stenotic dilatation to begin farther along in the ascending aorta.

The heart was within normal limits or very slightly enlarged as regards over-all size, but the configuration was abnormal. In all of our patients, the configuration of the left ventricular salient was suggestive of hypertrophy of the left ventricle. The left atrium appeared to be slightly enlarged in some patients, but this was not a consistent finding. No enlargement of the right atrium or right ventricle was observed and, although looked for carefully, there was no evidence of calcification in the area of the aortic valve in any of these patients.

Ancillary Investigations.—Ancillary measures have been resorted to but infrequently in the present group of patients and, in general, we have not considered that they offer anything in the establishment of the diagnosis. Direct pressure tracings from a peripheral artery or from the aorta always revealed changes reflecting the obstruction to outflow from the left ventricle into the aorta. In patients with less severe stenosis, this tracing may be normal. Brofman and Feil[20] considered evaluation of the recorded arterial pulse tracing as helpful in the differential diagnosis between valvular and subaortic stenosis. However, most other observers do not agree,[5, 27, 29] and it has been our experience that peripheral artery pulse curves are of no value in making this differential. Thus, while the peripheral arterial tracing reveals findings reflecting the stenosis, it is not a sensitive indication of the severity of the stenosis and in no way differentiates subvalvular from valvular stenosis.

Right heart cardiac catheterization is not considered to be of any value in the diagnosis of aortic obstruction. The cardiac output and pressures within the right heart are within normal limits in most instances. Downing,[4] however, has reported the finding of pulmonary hypertension in several instances. The left atrial pressure is frequently elevated in severe aortic obstruction. Retrograde transmission of the elevated left atrial pressure across the capillary bed might elevate the pulmonary artery pressure, or it is conceivable that, in certain susceptible individuals, the elevated pulmonary venous pressure might give rise to reflex mechanisms that would further elevate the pulmonary artery resistance and pressure.

Left heart catheterization would, of course, offer the most valuable information. The simultaneous recording of pressure within the left ventricle and aorta and the determination of the gradient across the aortic obstruction, when considered with the cardiac output at that time, would reveal rather accurately the severity of the obstruction. However, most of our patients have been children, the left atrium has been of normal size or but slightly enlarged, and it has not been considered advisable to perform left heart catheterization in these patients. Thus, while this would be valuable information to obtain pre- and postoperatively, we have not considered that left heart catheterization is such a benign procedure as to warrant its use in these patients.

SURGICAL CONSIDERATIONS

The avoidance of coronary air embolism, critical to the success of open transaortic approach to the aortic valve, depends first on the fact that exposure of the coronary ostia to air with a beating heart does not tend to drive air into the coronary arteries and, second, upon the development of effective methods to displace all air from the heart and aorta at the time of retreat from the heart. Our experience in this group of patients concerning the first factor has amply confirmed the experimental observations previously reported.[10] There is no tendency for air to enter the coronary system when the valve is exposed to atmospheric pressure.

As regards the second factor, many of the elaborate precautions taken in the earlier patients have not seemed necessary as experience has increased, so that the technique has been modified in the interest of simplification. Dissection, mobilization, and clamping of the lung roots, the right coronary artery, and the great vessels of the arch of the aorta have all been omitted as not apparently essential in the prevention of air embolism. Only one patient in this series had air enter the right coronary arteries and this was easily managed by finger massage and temporary cross-clamping of the aorta.

The current technique, therefore, is briefly described as follows. The patient is rendered hypothermic by surface immersion[26] and the lowest rectal temperature sought is 30° C. The transverse bilateral sternal-splitting incision is in the third interspace. The patient is positioned with the head of the table elevated and turned 20 degrees toward the right side. The venae cavae are mobilized and tapes placed around them. The aorta is carefully dissected downward at its base, mobilizing the fat pad in the groove between aorta and right ventricle and auricle until the myocardial portion of the aorta is reached. The right coronary artery is carefully observed and protected A flap of pericardium and adventitia, hinged laterally, is now raised over the proposed site of incision in the aorta, which should be in the most proximal portion of the aorta possible. Four stay sutures of 5-0 arterial silk are now appropriately placed in the aorta and a specially designed noncrushing clamp is applied (Fig. 3). This maneuver may be facilitated by temporarily occluding the venae cavae so that arterial blood pressure is momentarily lowered during the careful application of the clamp. The bite should be big enough to provide adequate tissue for incision, but not so great as to occlude blood flow in the aorta or obstruct the lumen of the ostium of the right coronary artery. The incision in aorta is made about 3 or 4 cm. in length.

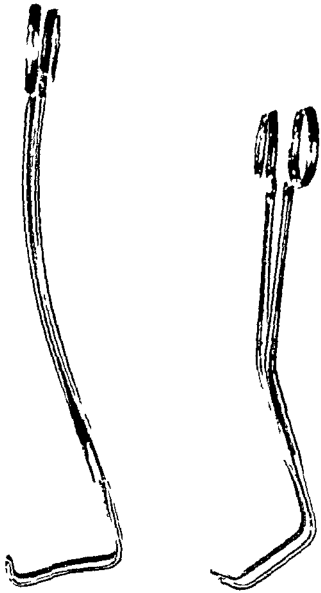


Fig 3.—Specially designed noncrushing clamps which have proved valuable for the aortic incision just above the valve The clamp on the left is made by George P Pilling and Son, Philadelphia, Pa, the one on the right by Bruno Richter, Lombard, Ill.

Inflow occlusion of the heart is now effected by traction on the caval tapes. A clamp is placed across aorta and 1.0 to 1.5 ml. of 1:4000 Prostigmin is injected proximally for coronary perfusion to slow the heart and protect against ventricular fibrillation. The transaortic clamp is removed. A noncrushing clamp is now placed across main pulmonary artery. The transaortic clamp is replaced after a few beats of the heart, and the clamp on the aortic incision is now removed. The aorta is usually entered about one minute after inflow occlusion has been achieved. After some blood loss as the heart empties the pulmonary veins, left auricle and ventricle, the field becomes completely dry.

The principle of the operation is based on the supposition that the thickened fused commissures can be accurately incised toward their insertion on the aorta in such a fashion that the cusps on both sides retain part of the commissural tissue and thus remain intact and competent. If no commissures are present, this principle could not be applied, without creating disastrous regurgitation. In this series of patients, as much incision as deemed possible was made in all three commissures (or in both commissures of a bicuspid valve). The operation on the valve itself, therefore, was maximal in terms of the underlying principle.

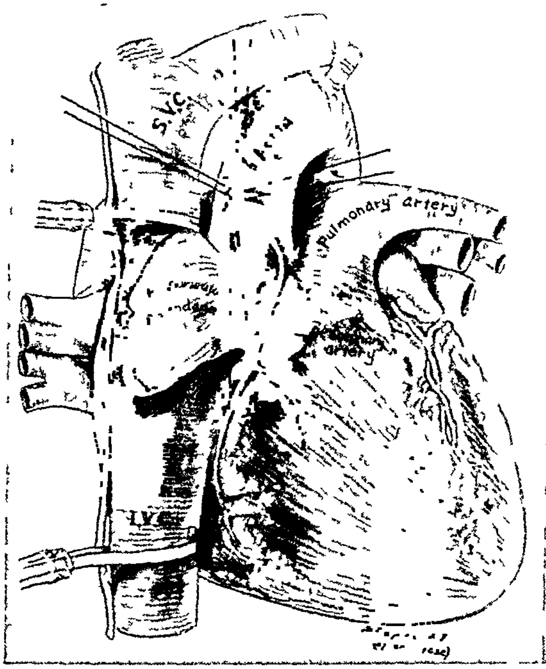


Fig. 4.

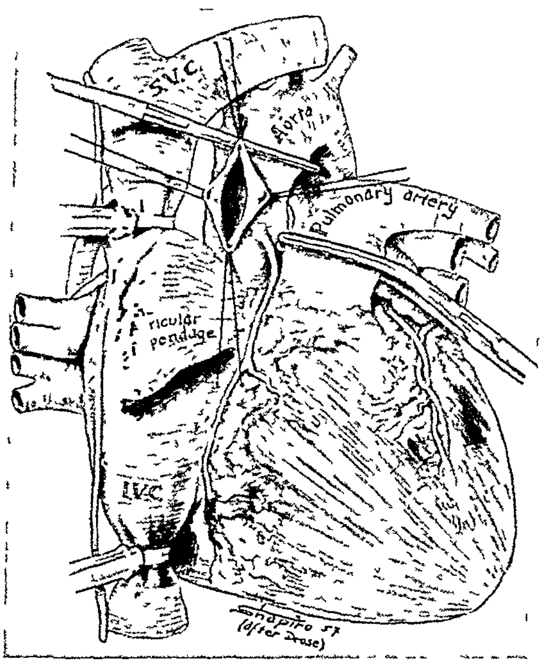


Fig. 5.

Fig. 4.—Following dissection of the root of the aorta, stay sutures are placed to facilitate the placement of the noncrushing clamp isolating the portion of the vessel which will contain the incision. Tapes have been placed on the venae cavae to provide inflow occlusion as needed. Right auricular tip is sutured laterally to auricle to provide exposure. The right coronary artery is exposed for protection during placement of the clamps.

Fig 5.—During the procedure on the valve, the inflow tapes are tightened, the aorta is clamped distal to the incision, and the pulmonary artery is also clamped. In the retreat from the heart, the left heart and aorta are allowed to fill with blood by removal of the pulmonary clamp and release of the superior vena caval tape. When all air has escaped, the noncrushing clamp is applied to the incision and the cross-clamp on aorta instantly removed. The inferior vena cava is then released to restore full circulation.

For subvalvular stenosis, the valve is held open by a suitable retractor. The constricting ring is identified and grasped with the forceps. The anterior and right lateral portions are excised with a knife or scissors. Great care must be taken to avoid injuring the mitral valve which lies posterior and slightly to the left, and which can be injured where the constricting ring is attached to its ventricular surface. Escape from the heart is achieved by removing the clamp from pulmonary artery and superior vena cava. Blood soon wells up in the aorta and when all air is thought to have escaped, the noncrushing clamp is carefully reapplied to the incision. The transaortic clamp is now immediately removed and blood flow is re-established. The tape on the inferior vena cava is released after an additional few moments. The total occlusion time varies between 5 and 7 minutes.

The aortic incision is now meticulously closed with figure of eight interrupted sutures, a Gelfoam pad is placed on the suture line, and held in place by resuturing the pericardial adventitial flap The aorta here is friable and does not sew as readily as arch or descending aorta The aortic closure is thus both difficult and nerve wracking

Pericardium is loosely closed, bilateral chest tubes are inserted for pleural drainage, the sternum is approximated with wire sutures, and the two thoracotomies closed with silk No dressing is applied to the incision During this portion of the procedure, the patient is being warmed with diathermy

RESULTS

The results of operation in this series are shown in Table I Of the 4 patients who had symptoms, the child with syncopal episodes prior to surgery as well as the one who complained of dizzy spells have had no symptoms since surgery and both are considered by their parents to have increased exercise tolerance The other 2 who had had diminution of exercise tolerance have shown very definite increase in activity Of the 4 patients who were thought to be asymptomatic prior to surgery, 3 remain so although one has had bouts of

TABLE I ELEVEN PATIENTS WITH CONGENITAL AORTIC STENOSIS TREATED WITH OPEN TRANSAORTIC OPERATION

PATIENTS	SEX	AGE	SYMPTOMS	RESULTS
			Valvular Stenosis	
E A	F	10	None	Bicuspid valve, thrill gone, murmur decreased
T M	M	5½	Episodes of syncope	Bicuspid valve, thrill gone, less murmur insufficiency minimal, no further syncope
J R	M	12½	None	Thrill gone, less murmur, moderate insufficiency, heart size unchanged, no symptoms
R F	M	12¾	Exertional dyspnea	Moderate insufficiency, febrile course, died of disruption aortic suture line on 27th day
C D	M	10	Exertional dyspnea, dizzy spells	Thrill gone, murmur decreased, all symptoms disappeared
M H	M	1 2/12	Excessive perspiration	Thrill gone, murmur decreased, moderate insufficiency, no change in heart size, no symptoms
C S	M	14	Decreased exercise tolerance	Thrill gone murmur decreased, minimal insufficiency, exercise tolerance increased
C E	M	5 9/12	Decreased exercise tolerance	Thrill gone, murmur decreased, exercise tolerance increased
			Subvalvular Stenosis	
W W	F	12 8/12	Decreased exercise tolerance	Thrill gone, murmur less, died 5th day, tracheobronchitis
C T	M	26	None	Thrill gone, murmur less, occasional auricular tachycardia
W B	M	5	Exertional dyspnea	Died day of operation

paroxysmal atrial tachycardia for which he refuses medical care. The other has been noted to have a great deal more energy than preoperatively and, in retrospect, it is felt that he actually did have some impairment of his exercise tolerance.

The systolic thrill palpable in all patients along the upper right sternal border preoperatively disappeared following surgery. Likewise, the systolic murmur, although it did not disappear, was universally decreased in intensity, quality, and in the extent of transmission.

Aortic insufficiency in varying degrees developed postoperatively in 5 patients, 2 of whom had a bicuspid valve, one of whom died of another cause. This is considered minimal in 2 and of moderate severity in 2. Increases in pulse pressure ranging from 20 to 57 mm. Hg were observed in these patients, reflecting both a decrease in the diastolic pressure as well as an increase in systolic pressure. On the other hand, those patients who had no murmur of aortic insufficiency showed a maximum increase of 20 mm. Hg in pulse pressure. Despite the increased pulse pressure in these 4 patients, there has been no evidence of cardiac enlargement or congestive failure. The end result of their insufficiency remains to be seen, but it appears to date that the benefit from removal of the obstruction outweighs the burden of the regurgitant flow.

Objective laboratory evidence of regression of left ventricular enlargement and post-stenotic dilatation has not been found. However, only a little more than a year has elapsed since the first patient of this series was operated on. The electrocardiogram, in all instances, shows no significant regression in the amplitude of the left ventricular complexes nor in the activation time in the left precordial leads. On fluoroscopy, there has been no evidence of increase in heart size, although the amplitude of the pulsations has been increased in all patients. This again may reflect the increase in cardiac output in some, but is probably indicative of the aortic insufficiency in others. There has been no apparent change in the degree of post-stenotic dilatation; however, there has not been any increase in the size of the aorta.

One patient (C. S.) developed fever, anterior chest pain of a pleuritic nature, and pain in the left shoulder 3 months postoperatively. There was no evidence of pleural or pericardial friction rub or fluid, nor was there evidence of an infectious process elsewhere. This was interpreted as the pleural pericardial syndrome. The patient responded promptly to a short course of steroid therapy and has had no recurrence of his symptoms.

Of the 11 patients, 3 died. One (W. B.) died several hours after surgery of acute and intractable congestive failure following removal of a subaortic lesion. The specimen revealed that the mitral valve had been injured in the resection of the constricting collar. One patient (W. W.) died on the fifth postoperative day of tracheobronchitis. She had had a subvalvular stenosis relieved and at post-mortem examination it appeared that most of the obstructing ring had been removed (see Fig. 1). There was a small patent ductus arteriosus present, hitherto unsuspected, and there was extensive bronchiolitis. A third patient

(R. F.) ran a protracted febrile course despite extensive antimicrobial therapy. Blood cultures were sterile but he died suddenly on the twenty-seventh postoperative day of hemorrhage from a pseudoaneurysm at the aortic suture line. He had had signs of aortic insufficiency postoperatively (see Fig. 2).

DISCUSSION

The differential diagnosis between aortic valve and subvalvular obstruction remains most difficult if not at times impossible. In the past it has not been considered that the clinical evaluation of the patient offers any findings that will allow this differential diagnosis. Some authors have held that the recorded arterial pulse tracing offers distinctive features, but most observers are not in agreement with this statement. At the present time, it is the opinion of most that ancillary investigations, such as left heart catheterization and angiocardiography, likewise will not enable this differential diagnosis to be established with any degree of certainty.

The factors that have been discussed in this paper are certainly not believed to be definitive in establishing anatomic diagnosis, however, we have found them helpful. Thus, the finding of the harsh, systolic murmur of maximum intensity in the third interspace, just to the left of the sternum or beneath the sternum, is considered to point toward the obstruction being at the subaortic level. The detection, where possible, of considerable dilatation in the very proximal portion of the aorta, that is, within the area of the sinuses of Valsalva, also points toward a subaortic stenosis. When both of these points are definitely noted, the diagnosis of subaortic stenosis is made. In this series, the diagnosis of subvalvular obstruction was made in 4 patients, and was correct in 3.

The establishment of etiology, especially in adults is also difficult. Unless the observer has personally followed the patient from the early years of life, he is not justified in stating that the lesion is of congenital origin. While it is possible that a mild congenital aortic valvular stenosis may, in the natural history of the disease, develop degenerative changes and calcifications, the vague history of a murmur in childhood by no means indicates that congenital aortic obstruction was the reason for the murmur. For this reason, it seems probable that many of the patients in the older age group recently reported to be of congenital origin,[3] in all probability had acquired forms of the disease.

In the past, aortic obstruction has been considered to be a relatively infrequent and benign lesion. However, recently it has been more frequently recognized. Those who have had the opportunity of following a series of these patients for a period of time have encountered sudden death as a not uncommon complication. The lesion can certainly not be considered benign. In our opinion, therefore, the finding of a clinical picture of significant aortic obstruction associated with definite and progressive hypertrophy of the left ventricle warrants surgical intervention.

As a result of our surgical experience in this series of patients, we are of the opinion that open operation upon the congenital aortic valve and subvalvular stenosis is technically feasible under hypothermia with circulatory occlusion.

Moreover, the pathology of the congenital obstructions is such that an excellent result can be achieved. *The operation has definite risks and should be applied,* at the present time, only in selected patients. The underlying theory that the pathology of congenital valvular obstruction will allow incision in the commissures with maintenance of competence of the cusps has been shown, we believe, to be generally true. However, in our application of this theory, aortic regurgitation has occurred sufficiently often to make us feel that the operator should err on the side of conservative relief of stenosis in order to prevent the undesirable occurrence of regurgitation. Aortic regurgitation unlike pulmonary regurgitation is a serious complication of surgery. Incisions into commissures, therefore, should be placed only in those commissures and only for such a distance that the operator feels quite confident that both adjacent cusps will remain competent. Most consistent results in the long run, therefore, will be achieved by the subvalvular type of obstruction, since in these patients, the risk of creating regurgitation does not exist and if care is taken not to injure the aortic leaflet of the mitral valve, relief of obstruction should be quite adequate.

Because of the precision required in the actual operative maneuvers for the correction of congenital aortic stenosis, it is our firm conviction that open direct vision technique is an essential component of the surgical therapy of this lesion.

SUMMARY

1. Eleven patients with congenital aortic obstruction were treated by direct vision transaortic surgical repair during circulatory arrest and hypothermia.

2. Eight of these patients had valvular and 3 had subvalvular stenosis. The pathology observed is discussed.

3. Recommendation for surgery depended on the clinical evidence of severe stenosis associated with left ventricular hypertrophy. Mild to severe symptomatology existed in 8 patients.

4. The results are encouraging but not uniformly ideal. The 8 surviving patients are considered to be improved, but slight to moderate aortic insufficiency exists in 4. Methods to prevent this complication in the future are discussed.

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(For Discussion, see page 190)

THE SURGICAL TREATMENT OF STENOTIC OR REGURGITANT LESIONS OF THE MITRAL AND AORTIC VALVES BY DIRECT VISION UTILIZING A PUMP-OXYGENATOR

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OUR steadily increasing experience with a pump-oxygenator for the curative treatment of various congenital intracardiac malformations over the past years has created confidence and assurance about the well-being of patients during an interval of total cardiopulmonary bypass.

As a direct consequence of this knowledge, the obvious benefits of comparable visual operations have been extended to acquired or congenital lesions involving the aortic and mitral valves.

The total number of patients operated upon for these ailments is as yet small and the surgical techniques to a variable extent are still in a developmental phase. Moreover, in these operations, as in other new fields of surgical endeavor, unexpected problems have been encountered and unanticipated pathologic variations have on occasion demanded on the spot improvisations which have not always succeeded initially. However, much has been learned through this inevitably painful method of trial and error and even upon the basis of this limited experience it has become obvious that direct vision offers improved therapy for patients with aortic stenosis or mitral stenosis, especially those with more advanced states of valvular destruction. Moreover, for the first time, a definitive corrective procedure for those patients suffering from mitral regurgitation appears possible, and a promise of a similar improvement in the functional accomplishments for those afflicted with aortic regurgitation is offered by these techniques.

Consequently more and more patients with these lesions are being managed by use of total cardiopulmonary bypass and open cardiotomy. The surgical techniques developed and the results obtained so far for these various types of valvular lesions are presented herein.

The Heart Lung Machine.—An integral part of success in these operations has been the visualization of the valves. Utilizing a pump-oxygenator and total cardiopulmonary bypass the surgeon can carefully perform the necessary reconstruction. Normal body temperature has been maintained during the

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surgery and this serves a dual function The incidence of cardiac arrhythmias is reduced and maintenance of the oxygenator blood at or a degree or two above the patient's body temperature effectively controls oxygen tension during oxygenation

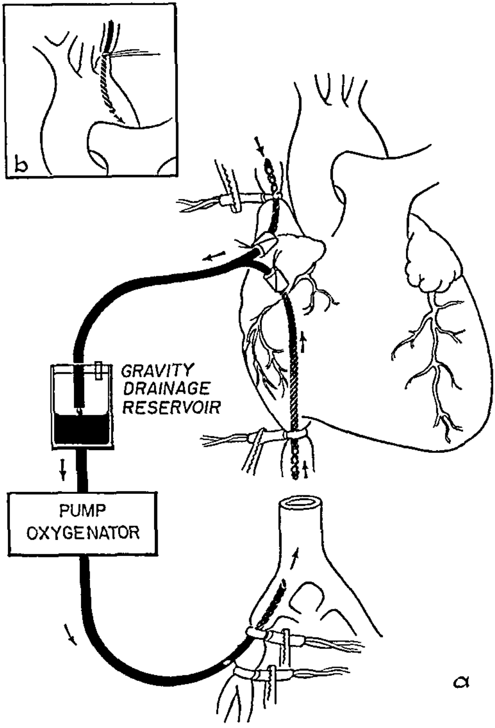

Fig 1—Technique of total cardiopulmonary bypass for open heart surgery The venous blood is drained by gravity siphonage For most patients the arterial blood is returned through a femoral artery catheter *Inset* Occasionally the subclavian artery is cannulated for this return

All patients were operated upon utilizing the helix reservoir bubble oxygenator[1, 4] This instrument* is constructed of disposable polyvinyl plastic tubing and actuated by a Sigmamotor pump† Our clinical experience with this pump oxygenator involves over 400 patients Despite its rather uncomplicated design, this apparatus has proved unsurpassed for the reliable and efficient maintenance of the cardiorespiratory functions for patients of any size

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*The helix reservoir bubble oxygenator components are made by The Phelan Manufacturing Company 2029 Washington Avenue South Minneapolis 4, Minn

†Sigmamotor Inc 3 North Main Street Middleport N Y

Perfusion Technique.—We prefer to drain blood from the venae cavae by gravity siphonage into a venous reservoir.[5] From this collecting chamber the blood flows or is pumped to the oxygenator. The arterial return is completed via a thin-walled plastic catheter* inserted into the subclavian or femoral artery (Fig. 1). During the bypass interval, certain physiologic checks are maintained over the patient by a monitoring electroencephalogram and a continuous measurement of the systemic blood pressure is obtained via an internal mammary artery catheter. The flow rate is adjusted to keep the electroencephalogram normal and the systemic blood pressure normal or near normal.[6] One of the distinct advantages of an occlusive pump over the nonocclusive design has been the greater ease with which it is possible to maintain a normal blood pressure in the perfused subject without the use of vasoconstrictor drugs during the bypass interval. Most adult patients will have a normal blood pressure at a flow rate of 50 to 75 c.c./Kg. of body weight with such a pump. Infants and smaller children may require perfusion rates in the range of 75 to 125 c.c./Kg. body weight per minute to achieve this goal.

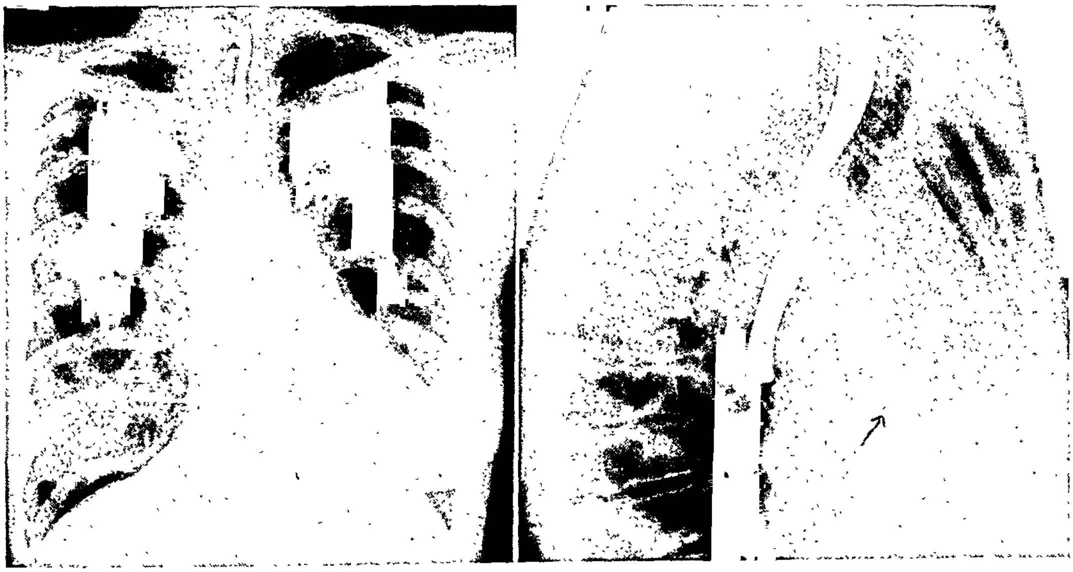

Fig. 2.—Calcific aortic stenosis. These preoperative roentgenograms are from the first patient (Case 1, Table I) operated upon utilizing the pump-oxygenator and retroperfusion of the coronary sinus. Note the heavy calcification in the aortic area evident in the lateral film.

When the size of the patient indicates that an oxygenating minute volume in the range of 2,500 to 5,000 c.c. may be needed, the double unit[4] is utilized together with the same pump (Sigmamotor TM-1).

AORTIC STENOSIS

Almost 2 years ago, in this clinic, a 39-year-old woman with severe acquired calcific aortic stenosis (Fig. 2) had a direct vision repair of the diseased valve utilizing a pump oxygenator in combination with retroperfusion of the coronary sinus.[7] This technique allowed for a precise, deliberate plastic repair of the valve while providing the contracting myocardium with oxygenated blood An excellent clinical result was achieved in this patient and she is living and working as a normal individual at this time. Since that initial case, retrograde perfusion of the coronary sinus has been utilized in 7 additional patients with lesions involving the aortic valve The results of these operations are presented at this time together with pertinent comparative observations derived from our experience in 9 other patients with aortic valve disease in whom alternative techniques (elective asystole with potassium citrate or acetylcholine, temporary coronary ischemia) for direct vision aortic valve surgery have been tested in conjunction with total cardiopulmonary bypass at normal body temperature utilizing the pump oxygenator.

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*C. R. Bard, Inc., Summit, N. J.

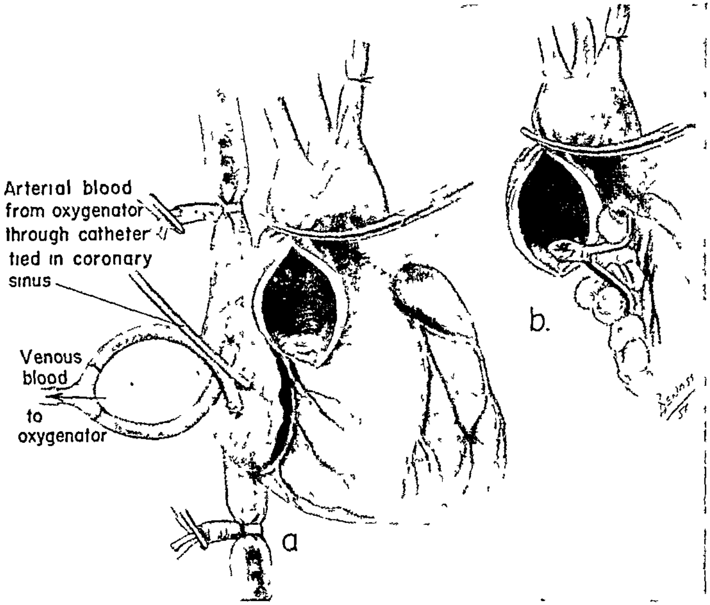

Fig 3—Technique for aortic valvuloplasty utilizing total cardiopulmonary bypass and retroperfusion of the coronary sinus to permit visualization of the aortic valve while maintaining the myocardial blood supply. b, The use of a heavy scissors or knife is sometimes necessary for valvotomy in the acquired lesions This is the safest method for aortic valve surgery

Method of Coronary Sinus Retroperfusion.—

The patient is connected to the pump-oxygenator in the usual manner (Fig 1) and the perfusion is begun, totally bypassing the heart and lungs The right atrium is opened and an appropriately sized plastic catheter (usually No. 14 to 16 Fr) with a rigid plastic tip is inserted into the coronary sinus and secured in place with a purse-string suture (Fig. 3). On the basis of considerable laboratory experience,[8, 9] it is believed that the optimal perfusing

TABLE I. PATIENTS HAVING RETROPERFUSION OF THE CORONARY SINUS DURING TOTAL CARDIOPULMONARY BYPASS FOR AORTIC VALVULOPLASTY

CASE AND AGE (YR.)	LESION	RETROPERFUSION TECHNIQUE	DURATION (MIN.)		OPERATIVE PROCEDURE	PRESSURES		CLINICAL RESULT
			RETROPERFUSION	TOTAL BYPASS		PREOP.	POSTOP.	
						AORTA L. VENT.	AORTA L. VENT.	
Case 1 39	Acquired calcific aortic stenosis	Separate retroperfusion pump (flow = 125 c.c./min.)	14	16	Created bicuspid valves because of obliterated cusps	—	—	Excellent
Case 2 28	Acquired calcific aortic stenosis	Separate retroperfusion pump (flow = 125 c.c./min.)	14	35	Commissurotomy	150/120 210/60	125/70 125/45	Died 2 days postop. from aortic insuff.
Case 3 52	Acquired calcific aortic regurg. and stenosis; mitral stenosis	Separate retroperfusion pump (flow = 125 c.c./min.)	13	18	Aortic valvuloplasty consisting of mobilization of leaflets by excision of synechiae in the sinuses of Valsalva and suture reconstruction; mitral commissurotomy	88/42 104/8	84/46 92/10	Excellent
Case 4 24	Congenital subvalvular stenosis	Y-connector in arterial limb	10	22	Incised the subvalvular diaphram	95/65 200/10	90/55 90/15	Excellent
Case 5 15	Congenital aortic valvular and subvalvular stenosis	Y-connector in arterial limb	32	45	Commissurotomy	80/15 175/15	—	Died, acute hemorrhage from tears in aorta
Case 6 5	Congenital aortic stenosis	Perfusion from reservoir at height of 50 cm.	2*	16	Commissurotomy	100/80 145/5	130/85 145/10	Excellent
Case 7 38	Acquired calcific aortic stenosis	Perfusion from reservoir at height of 50 cm.	2†	27	Commissurotomy	—	—	Excellent
Case 8 7 mo.	Congenital aortic stenosis	Perfusion from reservoir at height of 50 cm.	7	28	Commissurotomy	—	—	Died 8 hr. postop.; severe fibroelastosis of left ventricle

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*The patient had 60 mg. of acetylcholine injected in retrograde catheter after retroperfusion started, then had 10 minutes of total ischemia with bradycardia of 8 beats/minute.

†The patient had 200 mg. of acetylcholine injected in retrograde catheter after retroperfusion started, then had 8 minutes of total ischemia with bradycardia of 30.

pressure for this retrograde coronary sinus flow is a mean of about 35 to 40 mm Hg. We have achieved this perfusion pressure by several techniques, but the simplest method has been to perfuse the coronary sinus from a reservoir of oxygenated blood approximately 50 cm above the heart. The blood volume in the reservoir is maintained by an inflow of arterial blood from the oxygenator (Fig 4). Promptly after starting the retroperfusion, the ascending aorta is cross clamped and, if an aortic valvuloplasty is to be performed, the ascending aorta is incised longitudinally. The diseased valve is then repaired under direct vision. When the valvuloplasty is completed, the aortotomy is reapproximated by application of a suitable vascular clamp and then carefully closed with a running silk suture. Any residual air within the aorta is expelled by the retrograde flow. As soon as the aortotomy has been closed, the retroperfusion is terminated and the cross clamp removed from the aorta, restoring forward coronary perfusion.

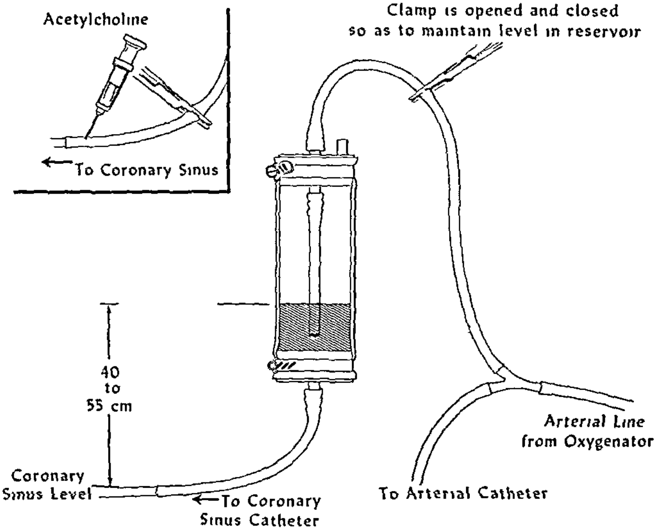
Acetylcholine
To Coronary Sinus
Clamp is opened and closed so as to maintain level in reservoir
40 to 55 cm
Coronary Sinus Level
To Coronary Sinus Catheter
To Arterial Catheter
Arterial Line from Oxygenator

Fig 4.—The most convenient method for controlling the pressure of the arterial blood retroperfusing the coronary sinus is to perfuse from a reservoir elevated 40 to 55 cm above the heart. *Inset*. Portrays one technique for retrograde arrest (see text for other details).

Recently (Table I, Cases 7, 8), we have used retrograde acetylcholine induced asystole in combination with retroperfusion. After commencing the perfusion of the coronary sinus acetylcholine is injected into the retrograde catheter until there is almost a complete standstill. The retrograde catheter is then clamped (Fig 4, inset). This technique facilitates the arrest in patients with some degree of aortic insufficiency and also provides a still, dry field during the period that the valvuloplasty is being performed. Before the aortotomy is closed, the clamp is removed from the retrograde catheter. This allows arterial blood to wash the acetylcholine from the myocardium and reinstitute good strong contractions before forward perfusion is restarted. In aortic valve lesions in which often some degree of insufficiency is present, it is very important that the left ventricle be beating at the time that forward coronary perfusion is restored otherwise irreversible left ventricular distention can occur. Two fatalities from this cause following potassium citrate asystole are summarized below.

An alternative method has been to add enough acetylcholine to the perfusing blood to maintain the arrest even though the retroperfusion continues. This procedure is deemed more feasible when a longer period of arrest might be needed such as in coronary artery surgery or aortic valvuloplasty for insufficiency.

Early in our clinical experience with retroperfusion, the coronary sinus was perfused with a separate Sigmamotor pump calibrated to deliver a predetermined flow of arterial blood to the myocardium. This technique was simplified subsequently by removing the pump and securing oxygenated blood directly from the main arterial limb of the oxygenator through a Y-connector. The pressure in this coronary sinus perfusing circuit was regulated by an adjustable clamp and monitored continuously by a strain gauge and Sanborn Twin-Viso recording apparatus. Although the two foregoing methods provided a proper coronary sinus flow, the technique of perfusing from a reservoir at 50 cm. has been the simplest and therefore the most satisfactory for clinical use (Fig. 4).

Surgical Technique for Aortic Valvotomy.—

Thoracotomy incision: The preferred incision for aortic commissurotomy under direct vision utilizing the pump-oxygenator has been a standard posterolateral thoracotomy through the bed of the right fourth rib with the patient in a straight lateral position and positioned on the operating table so that the kidney rest lies directly under the incision. Elevation of this rest after the chest has been opened gives excellent exposure to the ascending aorta. The caval cannulations and access to the coronary sinus for retroperfusion are satisfactorily accomplished with this approach. The arterial cannula has been inserted either into the right subclavian or common femoral arteries. The femoral arteriotomy is always repaired, and the subclavian arteriotomy is repaired in older children and adults.

The first patient operated upon by us for aortic stenosis, utilizing the pump-oxygenator for total cardiopulmonary bypass, had a bilateral anterior thoracotomy, but the exposure of the aortic valve was not improved by this incision over that obtained with the unilateral (right) thoracotomy and the likelihood of respiratory complications from the more extensive bilateral incision has been greater.

In several of the patients with congenital aortic stenosis where there was evidence of possible associated defects such as patent ductus arteriosus, pulmonary stenosis, or left superior vena cava, we have continued utilization of the bilateral thoracotomy to allow management of these associated lesions at the same time.

Aortotomy: After investigating several types of incisions in the aorta, we believe the most feasible to be a longitudinal hockey stick aortotomy which curves posteriorly as the annulus is approached and thus ends up almost parallel with the annulus and just above it. This incision prevents extensions into the coronary arteries by inadvertent tears and also the flaplike opening thus provided improves the visualization of the aortic valve cusps.

Clinical results with coronary retroperfusion for aortic valvuloplasty: Eight patients had aortic valve disease. Three of these individuals had congenital valvular stenosis, one had a congenital subvalvular stenosis only, and in the remaining 4 patients the aortic valve was the site of acquired disease. The pertinent data upon these patients are listed in Table I.

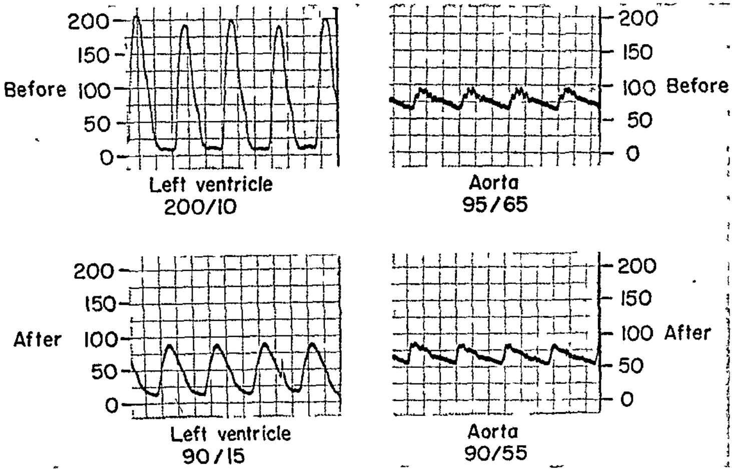

Fig. 5.—Subvalvular aortic stenosis (W. M., aged 24 years). The pressures measured just before and after direct vision excision of a subvalvular diaphragm through an open aortotomy. The aortic valve was normal (Case 4, Table I).

In the congenital valvular lesions, although the cusps were indeterminate in number, the leaflets were in all instances soft and pliable allowing a precise commissurotomy under direct vision. In all congenital valvular stenoses we have converted the valve opening into a bicuspid orifice. All the valves in the acquired disease group were heavily laden with calcium, which in some cases virtually obliterated the cusp commissure landmarks. Three of these had severe stenosis and the fourth had a predominant regurgitation with the valve fixed in a three-quarters open position. These valves were examined thoroughly at the time of surgery and if the commissures could be delineated they were incised carefully using either a scalpel or scissors. If the commissures were completely obliterated, two opposing incisions were made, thus forming a bicuspid valve which seemed to function very well (Case 1, Table I). Occasionally calcified spicules acting as struts between the cusp margins and the intima of the aorta within the sinuses of Valsalva were found and removed. In all cases dilators or the surgeon's fingers were passed through the valve to ensure an adequate orifice and to aid in mobilizing the leaflets following the placement of these incisions. Pressures were recorded in the left ventricle and aorta before and immediately after valvuloplasty in 4 of the patients and these are listed in Table I. The improvement in the respective pressure gradients after valvuloplasty is apparent from this data. One of the optimal results was achieved in a patient (Case 4, Table I) who had a subvalvular diaphragm. A wide incision of this diaphragm completely abolished the 100 mm. Hg pressure gradient between the ventricle and aorta (Fig. 5).

Four of the 7 patients with aortic stenosis have shown excellent clinical improvement as a result of direct vision valvuloplasty and are living and well at this time. Likewise, the patient (Case 3, Table I) with predominant aortic regurgitation has achieved and maintained an excellent clinical improvement following direct vision mobilization and reconstruction of the aortic leaflets.

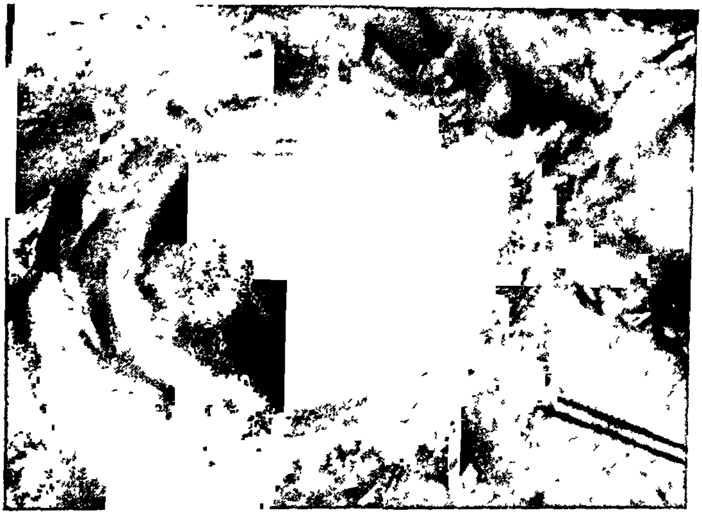

Fig. 6.—Patient with calcific aortic stenosis dying postoperatively of surgically induced regurgitation (Case 2, Table I) ; *for such a rigid calcified valve, stenosis can be relieved with a greatly lessened risk of regurgitation by creating a wide bicuspid rather than a tricuspid aperture.*

There were 3 deaths in this group of 8 aortic valvuloplasties. One patient (Case 2) had a severely calcified aortic valve and one of the commissural incisions created an intolerable insufficiency which contributed to his death 2 days after surgery (Fig. 6). Were we to operate now upon a patient with such a severely scarred and calcified valve in the light of more experience we would not attempt to restore a triangular orifice in such a case but rather create a wide bicuspid aperture which would relieve the obstruction without adding the considerable risk of aortic insufficiency.

A 15-year-old boy (Case 7) sustained an unfortunate complication when a transverse aortotomy just above the coronary arteries was utilized. During dilatation and excision of the subvalvular component of his stenosis, this circular incision was torn, completely transecting the ascending aorta. The aorta was reanastomosed during 32 minutes of retroperfusion and the heart continued to beat very well. Unfortunately 5 minutes of cardiac inflow stasis were required after termination of the retroperfusion to complete the repair of leaks in the posterior suture line of the aorta and the combination of this anoxia together with hemorrhage caused the heart to go into irreversible ventricular fibrillation. This transverse aortotomy is not now recommended. The third death was in a critically ill infant (Case 8) who had a congenitally stenotic valve opened widely under direct vision. The operative procedure and immediate postoperative interval were uncomplicated. There was no postoperative aortic insufficiency. She died suddenly 8 hours after surgery and post mortem revealed severe endocardial fibroelastosis of the left ventricle which probably contributed importantly to her death.

Retroperfusion of the coronary sinus has also been utilized for the repair of three other types of cardiac lesions: complete transposition of the great vessels,[8] aortic pulmonary septal defect,[8] and ruptured aneurysms of the sinuses of Valsalva.[10]

Other Techniques for Exposure of Aortic Valve Area.—

At the present time there are several techniques available to the cardiac surgeon which permit direct vision surgery in the area of the aortic valve and ascending aorta.

Hypothermia. Hypothermia which has been used by Lewis[11] and Swan[12] and their associates appears to us to have two significant disadvantages, namely, a limited operative time in this area and increased irritability of the myocardium. Therefore, we have not utilized this technique for aortic valve surgery.

Potassium citrate asystole. In this clinic, retroperfusion of the coronary sinus in combination with a pump oxygenator was first used on a patient in January, 1956, at which time no other oxygenator technique was available for exposure of the aortic valve. Retroperfusion has worked very well, but does entail the additional cannulation and purse string suturing* of the catheter into the coronary sinus ostium. Thus, when the cardioplegic drugs became available and proved satisfactory for other types of intracardiac defects,[13-15] such as ventricular septal defects and tetralogy of Fallot, we felt that cardioplegia could be the answer to a simplified method for temporary aortic valve exposure. Our initial experience with cardioplegia for aortic valve disease was with potassium citrate. Both patients died on the operating table due to our inability to restart their hearts after only very brief periods of asystole. Summaries of these two chastening experiences with complete cardiac arrest in patients with advanced aortic valvular disease appear below.

CASE A. The recent symptoms in a 38 year old man, leading to his referral for corrective surgery, were inability to work because of dyspnea and decreased physical strength associated with episodes of syncope upon mild exertion. A systolic murmur over the aortic area was first detected in 1946, while he was still in the Army. In 1952, he was hospitalized for this disability for the first time. In 1954, he developed subacute bacterial endocarditis which was successfully treated during a prolonged interval of hospitalization.

Physical examination disclosed a palpable thrill in the aortic area with a loud blowing systolic murmur of maximal intensity over the aortic valve area radiating up into the neck and in all directions. There was also a blowing diastolic murmur audible in the aortic area and transmitted down along the left sternal border of the sternum. The blood pressure was between 140/60 to 160/80 mm Hg at various examinations.

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*The use of a catheter equipped with a small Foley balloon makes the purse string suturing unnecessary and is therefore simpler technically.

Roentgenography showed marked cardiomegaly due to left ventricular enlargement, and calcification of the aortic valve area was readily apparent. The electrocardiogram was abnormal due to marked left ventricular hypertrophy and strain.

To-and-fro murmurs and these other findings confirmed the diagnosis of calcific aortic stenosis and insufficiency.

Operation was undertaken March 27, 1957, by entering the chest through the bed of the right fourth rib with the patient in a lateral position. The ascending aorta was huge in size and a significant systolic thrill was present in it. There was also a marked diastolic thrill in the left ventricle.

The patient was linked to the pump-oxygenator in the usual fashion, bypassing the heart and lungs. The aorta was cross-clamped proximal to the innominate artery and 120 c.c. of 2½ per cent potassium citrate injected into the first portion of the aorta produced asystole. The ascending aorta was then opened, disclosing one normal commissure and two fused ones resulting in a valve orifice of 12 to 14 mm. in diameter. The exposure was excellent. The two fused cusps were carefully incised to the aortic annulus and the leaflets mobilized, resulting in what appeared to be an excellent valvuloplasty. Following this, the aortotomy was closed and the aortic cross-clamp removed after an arrest period totaling 11 minutes.

The left ventricle remained in complete arrest and was absolutely resistant to all attempts to restart it. Sequentially, massage, Adrenalin, calcium, and electrical stimulation were utilized without avail. The left ventricle continued to redistend unless emptied manually. After 90 minutes of perfusion, during which these efforts were continued, the pump-oxygenator was turned off.

Autopsy disclosed an excellent commissurotomy. Massive left ventricular hypertrophy was present. An incidental finding of interest was a functioning pheochromocytoma of the right adrenal gland.

CASE B: This patient was an 8-year-old boy who had had a heart murmur, known since 14 months of age. Cardiac catheterization and exploratory thoracotomy for a patent ductus arteriosus was carried out elsewhere at the age of 5 years. No ductus was found and the pericardium was opened and a very pronounced thrill was found in the right ventricle. The aorta was enlarged, actively pulsating and, together with the continuous thrill, resulted in the diagnosis of ruptured aneurysm of the sinus of Valsalva. The operative procedure was then terminated. Subsequent recatheterization and angiocardiography were interpreted as confirmatory of this diagnosis.

Physical findings at the time of admission were a blood pressure of 108/0 mm. Hg, a palpable thrill over the precordium, and a loud continuous murmur heard over the sternum and transmitted to the right and left and to the back.

On April 29, 1957, corrective surgery was carried out. Upon opening the pericardium a marked thrill was present in the right ventricle as previously described. This thrill appeared to originate from the aorta in the region of the right coronary artery origin and this fact, together with some aneurysmal dilatation in this region, led to the diagnosis of a ruptured aneurysm of the sinus of Valsalva. The aorta was much enlarged contrasted to the usual case of ventricular defect and this too seemed to be in favor of a ruptured sinus of Valsalva aneurysm.

After insertion of caval and arterial catheters, bypassing the heart in the usual fashion, the ascending aorta was cross-clamped just below the innominate artery and 50 c.c. of 2½ per cent solution of potassium citrate diluted in blood was injected into the proximal aorta. This resulted in a prompt asystole and a right ventriculotomy was carried out disclosing, instead of the anticipated ruptured aneurysm, a moderately sized (1.5 cm.) membranous defect in the ventricular septum with complete incompetence of the aortic valve. Sutures were placed across the ventricular defect and several were tied. Before completing tying the remainder, the clamp was removed from the aorta in order to re-establish the cardiac beat after a period of asystole of only 4 minutes. However, throughout the remainder of the perfusion lasting 2 hours, no left ventricular beat could be re-established.

Whenever an attempt was made to try to close the defect or close the right ventricle, the left ventricle would enlarge tremendously. To control this complication, a tourniquet was even passed around the aortic annulus and beneath the coronary arteries to allow coronary perfusion and to prevent left ventricular distention. This was without effect upon the completely paralyzed left ventricle. Eventually the ascending aorta was opened and an attempt was made to repair the aortic insufficiency by direct suture of the leaflets converting them into a bicuspid orifice. This was successful in controlling the regurgitation but it remained impossible to restore a heartbeat. However, the approach used in this patient for the management of the aortic insufficiency developed under the duress of necessity did contribute to the realization that direct surgical treatment of aortic regurgitation through valvular reconstruction was feasible.

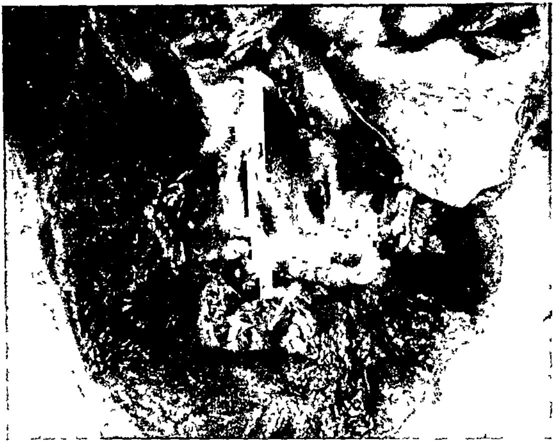

Fig 7.—Close-up of the left ventricle of an infant (12 mo of age) dying of congenital valvular aortic stenosis Note the massive hypertrophy that has occurred during this brief period The small size of the ventricular cavity also emphasizes one of the reasons these hearts are intolerant of a surgically induced aortic insufficiency.

Pathologic physiology: In advanced aortic valvular disease, the left ventricular hypertrophy is massive (Fig. 7) and doubtless exceeds the available coronary supply even though the latter may be entirely free of arteriosclerotic disease. This relative coronary insufficiency is the basis for the sudden deaths due to ventricular fibrillation characteristic of these patients. Moreover, it is not uncommon for the myocardium from hearts with advanced left ventricular hypertrophy to exhibit areas of ischemic necrosis. This latter finding is particularly liable to be present in the inner layers of the myocardium where the high left ventricular pressures constitute an added obstacle to the coronary circulation. If, in addition, there is superimposed some coronary artery obstructive disease as often happens in older patients with acquired lesions, it is obvious that the blood supply to these hearts often can be submarginal.

In such hearts it is understandable why potassium citrate, which gives a profound degree of relaxation of the cardiac musculature when perfused into the coronary circulation, is so dangerous. The problem appears to be primarily, although perhaps not entirely, a mechanical one, namely inability to wash out the potassium citrate even though, as in these 2 cases, the perfusing head of pressure is raised to above normal levels.

After these 2 tragic experiences with potassium citrate, we have abandoned its use for aortic valve surgery. These 2 patients both had advanced aortic valvular disease with marked left ventricular myocardial hypertrophy. One patient had only 4 minutes and the other 11 minutes of arrest. In both instances, when the aortic clamp was removed restoring coronary perfusion to the paralyzed myocardium, the flaccid left ventricle distended severely, augmented by the slight degree of aortic insufficiency which is present in many if not most of these cases. The severe distention of an ischemic flaccid heart in combination with a poorly perfused hypertrophied myocardium led to irreversible fibrillation in both cases. Moreover, effective massage is usually difficult because of the massive concentric hypertrophy of these hearts. After these 2 poignant experiences with complete cardiac arrest in cases with advanced left ventricular disease, it was realized that ideally the heart should be beating during the valvuloplasty or before systemic blood pressure was restored to the aortic valve. The contracting left ventricle could then eject any blood that might flow back through slightly regurgitant valves when the clamp on the ascending aorta was removed.

Case B illustrates another lesson worthy of emphasis; namely, the inadvisability of arresting any heart before opening it to inspect the nature of the pathology. To date, we have utilized induced cardioplegia in over 100 patients and this patient is the only one in the series in which we have failed to observe this precaution. In this particular patient, we were misled by the fact that the external pathology appeared so typical of ruptured aneurysm of a sinus of Valsalva, together with the fact that potassium citrate asystole had worked well in 2 patients[10] with this pathology.

Total cardiac ischemia: Since aortic commissurotomy, in some patients at least, can be carried out under direct vision quite adequately within only a few minutes of actual working time, the possibility occurred to us early in this work that one might simply ignore the myocardial oxygen requirements of the bypassed heart for this brief period.

The theoretical attractiveness of this approach has been further enhanced by the many recent experimental studies and a number of human observations indicating that the heart is surprisingly tolerant to anoxia under these circumstances. In a recent patient, documented elsewhere,[15] we observed the heart to recover sinus rhythm spontaneously after 35 minutes of total ischemia without cardioplegia. Moreover, convalescence was uncomplicated by any arrhythmias. However, this patient, as do the experimental animals on which much of this information is based, initially had a good myocardial reserve.

This is obviously not true of patients with aortic valve disease. Thus, we have not been completely attracted, as previously stated,[7] to this approach of elective anoxia. However, we did try this method in just one 49-year-old woman with acquired calcific aortic stenosis. Commissurotomy was carried out utilizing total cardiopulmonary bypass during only 5 minutes of complete myocardial ischemia at normal temperature The heart continued to beat during this time and no cardioplegic drug was used Despite the very short period of ischemia and an excellent commissurotomy, as determined by pressure determinations at surgery and confirmed later at autopsy, this patient died suddenly 36 hours after surgery and exhibited, at post mortem, extensive necrosis of the inner one third of the ventricular myocardium[16]

Use of acetylcholine asystole We have had experience with 6 patients with aortic valve disease utilizing total cardiopulmonary bypass at normal temperature together with acetylcholine induced asystole (Table II)

TABLE II CORRECTION OF AORTIC STENOSIS UTILIZING TOTAL CARDIOPULMONARY BYPASS WITH ACETYLCHOLINE* ASYSTOLE

CASE AND AGE (YR)	TYPE OF STENOSIS	MINUTES DURATION OF		ARRHYTHMIAS	RESULT
		TOTAL BYPASS	ARREST		
Case 1 5	Congenital valvular	12	3	Ventricular fibrillation†	Excellent
Case 2 22	Congenital valvular	20	7	None	Excellent
Case 3 11	Congenital subvalvular	12½	7	Ventricular fibrillation†	Excellent
Case 4 6 mo	Congenital valvular	13	5	Cardiac arrest 6 hr postop , good immediate recovery	Died 48 hr postop , no clear cut cause of death debilitated preop
Case 5 7	Congenital valvular	17	6	Ventricular fibrillation†	Excellent
Case 6 49	Calcific aortic stenosis	62	53	Ventricular fibrillation†	Severe aortic insufficiency created by initial commissurotomy which then was corrected by valvuloplasty Died 2½ days postop of left ventricular failure due to myocardial necrosis

*10 mg/Kg body weight

†Developed shortly after restoration of coronary arterial flow easily defibrillated with electric shock and pump oxygenator support

In general, this experience has been quite satisfactory and certainly if cardioplegia alone is to be used in these hearts with massive left ventricular hypertrophy acetylcholine appears to be somewhat safer than potassium citrate

Acetylcholine often does not completely arrest the heart immediately and allows it to beat slowly for a variable period during the cardiac ischemia before complete asystole occurs However, the myocardium in the acetylcholine arrested heart is very responsive to external stimuli such as massage and thus the beat may be quickly restored This obviates the very dangerous complication of ventricular distention encountered with the profound relaxation of potassium citrate arrest The technique of using acetylcholine is not without some danger, however, for this slowly beating ischemic heart may be lizing more energy than the completely stilled heart. The much greater incidence of ventricular fibrillation in these hearts (Table II) in comparison to those with retrograde coronary perfusion is perhaps significant in this regard. For these reasons, the period of ischemia utilizing acetylcholine should be brief. It does appear from the experience in this small clinical series that acetylcholine cardioplegia can be used safely for aortic stenosis if this period of ischemia is no longer than 10 to 15 minutes, the myocardium is not greatly hypertrophied, and the cardiac reserve reasonably good.

However, Case 6, Table II, illustrates the fact that it is often not possible to be certain a priori of the duration of any intracardiac procedure. In this man the initial commissurotomy resulted in an obvious aortic insufficiency. In the subsequent interval, it was possible to obtain an excellent reparative result upon the valve preserving an adequate orifice and correcting the insufficiency (Fig. 9) but the cardiac ischemia was severe. It is likely that retrograde perfusion during this interval might have made the difference between success and failure in this case.

Current Procedure for Aortic Valve Lesions.—

At the present time we are using total cardiopulmonary bypass together with acetylcholine asystole for the patients with congenital aortic stenosis who as a group are much better operative risks.

In all patients with acquired aortic lesions and in those with congenital lesions in whom the valve leaflets are severely diseased, the ventricular myocardium is greatly hypertrophied, and a longer period is contemplated for the valvuloplasty, we believe retrograde perfusion of the coronary sinus as an adjunct to the cardiopulmonary bypass is the safest procedure.

AORTIC REGURGITATION

Insufficiency of the aortic valve has presented a formidable challenge to surgical treatment. The palliation afforded to certain far-advanced cases by insertion of a Hufnagel valve[17] into the thoracic aorta has been of definite value. However, quite obviously something more curative is necessary.

The thought has been advanced by some that nothing can be done for this lesion until a completely satisfactory prosthetic valve is available and one which can be inserted so as to replace the diseased valve and lie proximal to the coronary artery ostia. We would disagree with this philosophy for at least two reasons. First, such a completely satisfactory valve is not likely to come about until a greater familiarity with the pathologic anatomy has been acquired by surgeons working within this area under direct vision. Second, if experience elsewhere can be taken as any guide, once this familiarity has been acquired it will be likely that methods for reconstruction of diseased valves will evolve and a completely artificial valve may not be needed nearly as often as current predictions suggest. This sequence has evolved recently in respect to the mitral valve.

Much of our small experience to date in regard to the development of a direct reparative procedure for aortic insufficiency has been an outgrowth of commissurotomy procedures for aortic stenosis in which a significant or obviously intolerable degree of insufficiency was produced inadvertently.

Pathologic Anatomy of Aortic Regurgitation.—

One very favorable anatomic aspect of the primarily aortic insufficient lesion that has impressed us, when these hearts were studied in the fresh state, was the suppleness of the leaflet tissue together with usual absence of calcium. This is in marked contrast to the usual pathologic change found in those patients with acquired aortic stenosis. There is a parallel here with the mitral valve where, in regurgitant lesions, the pathologic alterations in the leaflets per se are frequently much less severe than those found in stenosis, yet the surgical treatment of regurgitant lesions has lagged far behind. The reasons for this are, of course, clear. Stenotic valvular lesions are often successfully widened by blind techniques whereas direct vision seems essential for a successful valvuloplasty for insufficiency. But beyond that, it is predictable that the results of surgical correction of insufficiency either of the mitral or aortic valves is likely ultimately to surpass that possible for stenotic lesions because of the better cusp or leaflet tissue remaining with which to work.

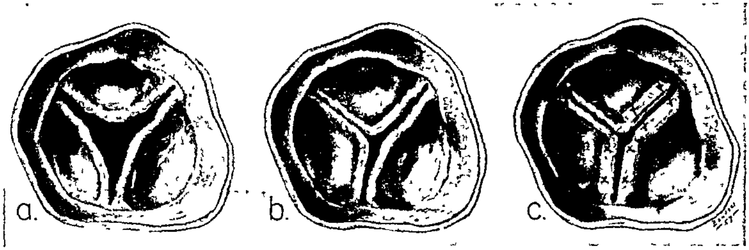

Fig 8.—Technique for the direct vision surgical correction of aortic insufficiency of the primary type.

a, The usual pathology is portrayed. There is a shrinkage of the cusps due to moderate thickening and retraction, but calcium is usually absent and the leaflets remain flexible. Annular dilatation, except in the syphilitic patients, is usually not prominent. As a consequence of these changes, coaptation of the cusps during diastole is imperfect.

b, Our concept of treatment is to obliterate one commissure entirely by silk sutures leaving a wide bicuspid opening.

c, Any actual loss of cusp substance or residual failure of the cusps to coapt completely is corrected by the addition to the leaflet margins of pledgets of compressed polyvinyl sponge (Ivalon) as needed.

This technique of repair is also applicable to the regurgitation that may follow procedures to relieve aortic stenosis.

In the nonsyphilitic patients with a rheumatic etiology for their aortic insufficiency, annular dilatation has not appeared to be a very significant element. Rather the leaflets, although often only slightly or moderately thickened, are still very pliable but fail to meet together tightly enough because of thickening and a tendency for the free edges of the cusp to roll or curl back (Fig. 8, *a*). These observations have constituted the basis for a valvuloplasty procedure described below.

A Surgical Technique for the Correction of Aortic Insufficiency.—

The concept that we have developed (and on the basis of our to-date limited experience it appears to have merit) is to convert by suture the normally tricuspid aortic valve into a bicuspid orifice and reinforce and give additional support if necessary to the lips of this bicuspid opening by suturing to its edges small pledgets of compressed polyvinyl sponge (Ivalon) (Figs. It is possible to convert the valve mouth to a bicuspid aperture by interrupted stitches of silk. This adds significant support to the leaflet tissue without the concomitant creation of stenosis. The addition of these Ivalon strips corrects the tissue deficiency resulting from the curled cusp margins and the added weight of the Ivalon also contributes to their tighter closure during diastole. A favorable observation noted at surgery in our patients is the fact that these slightly or moderately thickened cusps hold fine silk stitches well and much better than would be true for the paper-thin normal cusps.

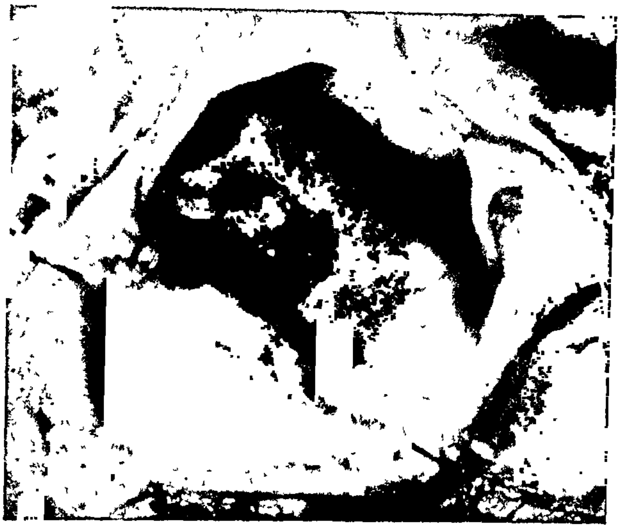

Fig. 9.—Aortic valve from patient in whom aortic valvuloplasty was carried out for a surgically induced aortic insufficiency (Case 6, Table II). In this man, operated upon for calcific aortic stenosis, the opening of all three commissures resulted in a severe regurgitation. This was corrected completely by the valvuloplasty procedure portrayed in Fig. 8, leaving a widely patent and competent aortic aperture. The Ivalon pledget sutured to the leaflet margin covers the suture line obliterating the third commissure (see text for details).

Report of Cases.—[*]

The clinical application of these observations is in the developmental stage at this time. Only 3 patients have had some attempt made at correction of a significant degree of aortic insufficiency. These presented concepts have evolved from our experience to date.

The first patient operated upon (Case 3, Table I) has been the only successful result to date. Progressive incapacitation due to fatigability and both right and left cardiac decompensation were the indications for surgical therapy in a 52-year-old lady whose operation was carried out on May 23, 1956, utilizing coronary retroperfusion in addition to total cardiopulmonary bypass. This patient's valve lesion was a mixture of mild stenosis and considerable regurgitation of rheumatic etiology. The cusp leaflets were fixed in an approximately three quarters open position, in part due to adhesions across the sinuses of Valsalva These were freed with the scissors hereby restoring considerable mobility to the leaflets Nevertheless, although some improvement had resulted, significant insufficiency still remained due to a failure of the leaflets to meet satisfactorily in the midline (this is demonstrated in Fig 8, *a*) Two mattress stitches were placed as in Fig 8, *b* with obvious improvement No polyvinyl sponge was utilized to supplement the area of the cusps in this patient The proximal aorta was considerably dilated Therefore in closing the aortotomy a generous elipse was excised from the aortic wall in order to bring about a "snugging up" effect upon the annulus

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[*] Since submission of this report the first patient with pure aortic insufficiency has been operated upon. O. B., aged 45 years, had had rheumatic fever at 6 years of age. He remained well until age 30 when the first symptoms of heart disease were manifested. Deterioration in the year immediately prior to surgery had been rapid with acute pulmonary edema upon effort, angina pectoris, and claudication, massive cardiomegaly, a loud blowing diastolic murmur in the aortic area, and a blood pressure of 220/50/0 mm. Hg.

Operation was undertaken Jan. 8, 1958, and the aortic valve pathology and its method of repair was nearly identical to that portrayed in Fig. 8. The interval of pump-oxygenator support was 48 minutes and the heart was retroperfused for 25 minutes without arrest or any arrhythmias, despite the very poor cardiac reserve. Convalescence was uncomplicated. The diastolic murmur and water-hammer pulse disappeared completely. His blood pressure fell immediately too and has remained at $\frac{120 - 130}{75 - 85}$ mm. Hg. All evidence suggests a complete correction of the regurgitant leak in this man.

Our second experience with this problem occurred in the child already described in Case B Potassium citrate arrest was used Preoperatively, the patient was thought to have a ruptured aneurysm of Valsalva Actually he had a ventricular septal defect with a grossly insufficient aortic valve Although this case terminated unsuccessfully, we were gratified by the degree of improvement induced in this very severe case of aortic regurgitation utilizing mattress stitches of silk placed again as shown in Fig 8, *b* This experience emphasized to us that a definite accomplishment in thwarting the aortic leak had been achieved

The third patient, reported in Table II as Case 6, was a 49 year old man with clinically far advanced disability due to calcific aortic stenosis This apparently had evolved after a severe, febrile illness in childhood

Right and left heart catheterization carried out 2 months prior to surgery disclosed the following pressures (mm Hg) left ventricle 180/28, femoral artery 104/51, and pulmonary artery 75/36 No pressure gradient existed across the mitral valve which denied the existence of significant disease at that site These studies supported the diagnosis of severe aortic stenosis with chronic left ventricular decompensation Calcium was plainly visible and in considerable quantities in the aortic valve area in his chest roentgenogram

Corrective surgery, utilizing a right thoracotomy and total cardiopulmonary bypass, was undertaken After cross clamping the ascending aorta and just prior to opening it, the heart was arrested with a proximal intra aortic injection of 500 mg of acetylcholine On exposure of this area we noted the valve was severely stenosed and had a rigid eccentric slitlike orifice, 4 or 5 mm in length, located at the one commissure The other two commissures had been completely fused by heavy deposits of calcium Vision and exposure were excellent We carefully incised the two obliterated commissures with the knife and scissors opening them widely, well out to the annulus These maneuvers restored the normal tricuspid aperture When the aortotomy was closed, the heartbeat came back promptly after restoration of coronary flow and the heart and lungs were bypassed for 21 minutes However, it was apparent at once that although good relief from the stenosis had been obtained, a very pronounced diastolic thrill now existed over the left ventricle Moreover, the heartbeat during the succeeding 5 to 10 minutes became much weaker and arrest appeared imminent Therefore the pump oxygenator was turned on and the cardiopulmonary bypass restored The aortotomy was reopened This time the valve was converted into a bicuspid one by sewing up one commissure completely with interrupted 3 0 silk mattress stitches as shown in Fig. 8, *b*. Again the aorta was closed after this second bypass interval of 18 minutes. The heart had gone into ventricular fibrillation and electrical shocks were required to restore rhythmic contractions. The diastolic thrill in the left ventricle seemed less evident to palpation but was definitely still present. In confirmation of this imbalance, the cardiac contractions became progressively more feeble. Therefore we elected to reopen the aorta once again. Our reappraisal painstakingly carried out revealed the cause of continuing insufficiency. These bicuspid leaflets which had been formed by the sutures were inadequately coapting due to the loss of substance and curling of the margins present. Compressed Ivalon (6 to 1) was fashioned in the form of a small pledget and sutured to the edge of the deficient leaflet using mattress stitches of silk (Fig. 9). To the eye this gave a far better correction of the valvular insufficiency. The heart had again gone into ventricular fibrillation during this third period of anoxic arrest of 14 minutes' duration and was once more defibrillated. This time it took over at once and established an excellent beat. The left ventricular diastolic thrill had completely disappeared. Significantly too, and in confirmation of this was the fact that the patient achieved and maintained a near normal blood pressure without pump-oxygenator support. For the second and third aortotomies no acetylcholine was used. Thus, the heart was in anoxic asystole during both of these intervals. The aorta was cross-clamped a total of 53 minutes and the total perfusion duration was 61 minutes at a perfusion rate of 3.3 L. per minute (55 c.c./Kg. per minute).

Postoperatively, despite this complicated procedure, his condition appeared surprisingly good. He awakened immediately, was alert, and had a normal systemic blood pressure. The pulse rate was only 80 to 100 per minute. The electrocardiogram was bizarre. It appeared to be made up entirely of ventricular extrasystoles. Because of the slow rate and good blood pressure no specific therapy was instituted for this arrhythmia. On the following morning the electrocardiogram seemed much improved revealing a return of sinus rhythm, and a disappearance of the ventricular extrasystoles. Other evidences of severe myocardial depression persisted, however.

His clinical progress during the next 2 days appeared to be quite uncomplicated until rather suddenly (within one hour) on the third postoperative day he developed progressive dyspnea, orthopnea, and pulmonary edema. Despite rapid digitalization and tracheotomy he died. Autopsy disclosed a gratifying reconstruction to the valve (Fig. 9). Both the stenosis and regurgitation seemed to have been corrected. Death was due to left ventricular failure secondary to widespread areas of focal necrosis in the myocardium.

Technical Considerations in Aortic Valve Reconstruction.—

These cases portray the development of some of our techniques for aortic valvuloplasty. While much undoubtedly remains to be learned we have been encouraged by the extent to which these diseased and distorted valves can be restored to function without dependence on a completely artificial valve which moreover is as yet unavailable in a workable and tested form. Several technical considerations derive from this experience and are deemed worthy of emphasis. First, in aortic stenotic valves of the acquired type which are severely distorted and rigid from scarring and calcification, the operator is naturally tempted to open all three commissures. This is unwise because of the likelihood of creating too much insufficiency. It seems preferable to relieve the valvular obstruction by limiting the commissuroplasty to establishment of a bicuspid aperture. Second, if necessary, any loss of substance can be replaced by suturing pledgets of compressed Ivalon or nonwettable Silastic plastic to the cusp edges. We believe ample evidence now exists from the congenital lesions to indicate that bicuspid valves will provide good function once the element of stenosis has been relieved. Our first patient with aortic stenosis who was operated upon under direct vision 2 years ago had a valve heavily laden with calcium. It was converted into a bicuspid valve of necessity. Not until later experiences did we realize the value and importance of this concept. To be sure for those stenotic lesions with lesser degrees of destruction the creation of a tricuspid orifice is probably preferable if it appears that the leaflets are supple enough to regain their function. This decision as to which type of procedure to try is best made after the valve is visualized. At all times it is reassuring to know that with the pump-oxygenator bypass technique considerable opportunity is provided for correcting any inadvertently induced aortic insufficiency, as well as for dealing with the many pathologic variations encountered, and even for insertion of an artificial valve.

These techniques cited above in the management of aortic insufficiency created by attempts at relieving aortic stenosis have definite applicability to the direct surgical approach to primary aortic insufficiency. In summary they are: creation of a wide bicuspid orifice by suturing one commissure and overcoming any loss of substance by addition of pledgets of compressed Ivalon to these cusp edges as portrayed in Fig. 8.

Others have suggested that aortic insufficiency can be managed by reducing the circumference of the annulus by circumferential plication or resection of a portion of the annulus in the noncoronary area. Although we rather doubt that either of these approaches will be commonly useful and may well create new technical problems we believe they, particularly resection of the annulus, should be kept in mind as supplementary measures.

MITRAL REGURGITATION

For almost 10 years now, stenotic lesions of the mitral valve have been satisfactorily treated by well-known surgical measures involving finger fracture supplemented in some patients by commissural incisions. However, the regurgitant lesions of the mitral valve, despite their clinical importance as a major cause of cardiac disability, have continued to challenge cardiac surgeons and numerous and diverse surgical approaches have been described. Many ingenious devices have been inserted into the mitral orifice in an attempt to diminish the regurgitant flow. Most of these techniques have been directed at the posterior commissure for this is the usual site of reflux; the anterior one is less frequently insufficient because of the greater reserve of leaflet tissue in that area.

Substances such as pericardial tubes,[18] vein grafts,[19] and tendon grafts[20] placed across the posterior leaflet to diminish the regurgitant channel have usually eventuated in shrunken fibrotic struts. A rigid Ivalon sponge,[21, 22] a mobile Ivalon sponge sling,[23] and a Lucite baffle[24] have also been placed in the mitral orifice but with limited success.

Approximation and elevation of the posterior aspect of the mitral valve have been attempted by the use of pericardial strips,[25] vein slings,[26] silk sutures,[27, 28] artificial cusps formed of nylon,[29] inverted veins,[30] and pericardium.[31]

Davila, Glover, and associates[32, 33] have advocated the placing of a circumferential suture around the mitral annulus to narrow the regurgitant area.

Present Approach.—

The large variety of operations proposed for the correction of mitral insufficiency confirms to some extent the general ineffectiveness of many of these techniques. Nevertheless, an analysis of these experimental and clinical endeavors has brought additions to our basic knowledge about this lesion. Several of the techniques reviewed above seem sound in theory. Perhaps therefore a major reason for their lack of uniform success arose from the obvious consequences of their having been done as blind procedures. Certainly the ability to observe the diseased valve during surgery should self evidently improve the surgical results by assuring that the particular reparative procedure be performed which fits the specific lesion being looked at functionally. The development of the pump-oxygenator as a safe and effective tool has now made such open operations upon the mitral valve both possible and feasible.

Pathologic Physiology of Mitral Regurgitation.—

Mitral insufficiency may result from varying combinations of two primary factors: (1) an absolute loss of valve substance from cicatricial retraction and shrinkage of leaflet tissue and/or chordae tendineae, and (2) dilatation of the annulus fibrosus. Furthermore, these two factors are interrelated to the extent that severe insufficiency may result from compensatory left ventricular dilatation secondary to an initial deficiency—not necessarily severe—in the valve substance.

Moreover, for this same reason, mitral insufficiency once established, tends inherently to progress. The regurgitant leak into the left atrium begets a compensatory left ventricular enlargement to maintain the cardiac output; and this, by further stretching of the annulus, produces an ever greater mitral leak. In confirmation of this is the clinical cardiologic observation that regurgitant lesions of the mitral valve are tolerated progressively less satisfactorily with time than are stenotic ones.

Surgical Concepts and Technique.—

The mitral valve has been visualized in all patients utilizing the helix reservoir bubble oxygenator and the Sigmamotor pump for the necessary interval of total cardiopulmonary bypass.

Elective asystole has not been utilized since it has appeared to be disadvantageous for these procedures to have the ventricles in repose in contrast to the experience in certain of the complex forms of congenital heart disease where it has been most helpful. In several of the patients there was a copious influx of blood into the operative field from an incompetent aortic valve We have managed this by intermittent aortic occlusion as previously described.[31]

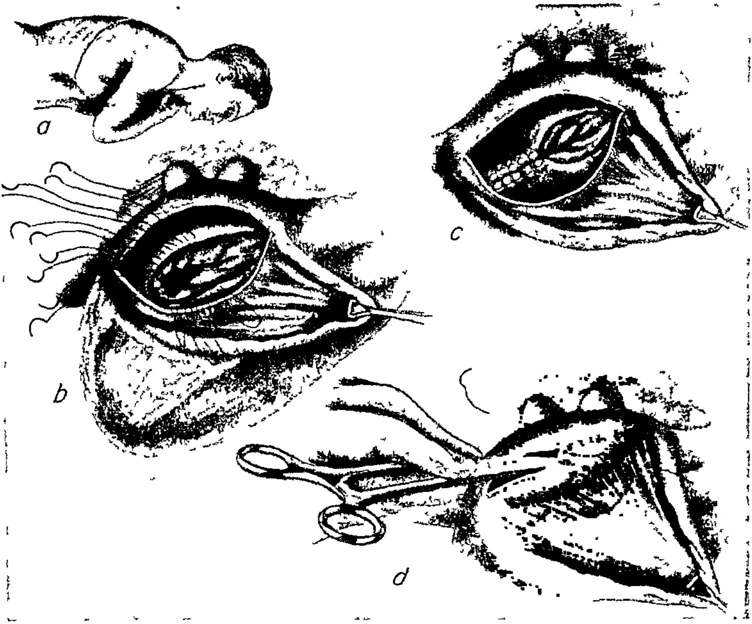

Fig 10—Correction of mitral regurgitation by direct vision annuloplasty

a, Standard posterolateral right thoracotomy incision through the bed of the fifth rib

b, Placement of mattress stitches of heavy silk in annulous fibrosus of incompetent portion

c, These stitches are tied tightly over pillows of compressed polyvinyl sponge to prevent any cutting out before the approximated annulus has healed securely.

d, After valve competency has been restored, the usual precautions against retained air in the left ventricle are necessary as the atriotomy is closed

Annuloplasty is utilized in those patients where the regurgitation is due in part or entirely to annular dilatation

Surgical incision: One of the important elements of success with all 5 of these patients has been the choice of a right posterolateral thoracotomy incision to approach the left atrium. Unquestionably the best exposure of the mitral valve is obtained through a left atriotomy performed from the right thorax. This anatomic consideration is more easily appreciated when it is recognized that the left atrium is actually posterior rather than sinister. With the patient in a right lateral position, the thoracotomy is performed through the bed of the right fifth rib. The venae cavae are cannulated in the usual manner through the right atrium. Through a purse-string suture, a third cannula is placed into the right atrium in order to aspirate the coronary sinus blood before it passes to the lungs and upon return obscures the operative area in the left atrium. The arterial cannula is usually placed in the right common femoral artery. After the perfusion is begun, the wall of the left atrium is incised longitudinally near the interatrial groove, anterior to the insertion of the pulmonary veins (Fig. 10). This opening is readily made without the need for any dissection of the interatrial sulcus, because the left atrium has been of giant proportions in these patients. This atrial incision provides excellent exposure of the entire mitral valve region. Under direct vision with the left heart essentially free of blood and with the left ventricle beating, the pathologic lesion can be carefully assessed by inspecting the leaflets, their commissures, and the chordae tendineae. Often, if the operative field is unusually dry due to a normal aortic valve we have found it helpful to temporarily clamp the catheter aspirating the coronary sinus blood. This provides enough blood to evaluate valve function. Any stenosis present at the commissures is evident and readily correctable with the scissors. Fused chordae tendineae, when present, can be separated. The area of insufficiency has been frequently located at the posterior pole (Cases 1, 2, 3), but was also found to arise anteriorly (Case 4) and even involve an entire leaflet (Case 5).

Method of annuloplasty: This technique of suturing the mitral annulus under direct vision with silk suture was first tested in the animal laboratory. The procedure was well tolerated despite the fact that the normal canine valve is an unusually delicate, paper-thin structure and thus a poor imitation of the diseased regurgitant valve in man. Clinically, when the annulus was greatly enlarged (Cases 1, 2, 3, 4), annuloplasty was performed above the insufficient area by placing interrupted mattress sutures of heavy silk (No. 2, with double-armed swedged-on needles) in the annulus fibrosus. These were tied down over pillows of compressed Ivalon sponge to prevent any possibility of their cutting out before healing was complete. These stitches approximate the annulus and hence reduce orificial circumference. Occasionally as many as seven or eight interrupted mattress sutures have been placed in order to eliminate the regurgitant jet. This beating heart with its tonic myocardium has been very helpful for evaluating the regurgitation tendency during the annuloplasty. The blood ejected by the contracting left ventricle continues to delineate the area of insufficiency and indicates when the operative repair is satisfactory. As the annulus is approximated, the surgeon may observe exactly how much additional plication is required for the best function. In each of the 4 patients with pathologic changes, characterized primarily by annular dilatation (Cases 1 to 4), it was possible to reduce the annular length enough to correct the regurgitation and still leave a mitral orifice of normal size (2 or 3 fingerbreadths), i.e., no element of stenosis was introduced by the surgical correction.

In placing these stitches in the annulus, generous bites of this tough tissue are taken and this fact together with their being tied over pledgets of Ivalon makes any late reopening, before healing has occurred, appear unlikely. In placing these stitches in the annulus from inside the left atrium, one is working close to the coronary artery directly opposite and just outside of the auricular wall. This technical consideration should be kept in mind so as to avoid any compromise of this vital vessel, particularly at the anterior pole. After annuloplasty is completed, the atriotomy is closed with a running everting silk suture, and the perfusion is terminated.

Insertion of prosthetic valve leaflet: In Case 5, despite a giant left atrium and advanced left ventricular enlargement, the annulus of the mitral valve was of normal circumference. The valve was markedly insufficient because of virtually complete disappearance of the mural (posterior) leaflet by cicatricial retraction. Several plicating mattress stitches of silk were placed in the posterior annulus, and tentative approximation of these produced stenosis, without correcting the regurgitation, and so they were removed. Clearly the abnormal leaflet had to be replaced by a space-occupying prosthesis which could provide sufficient substance and against which the flexible aortic (anterior) leaflet could coapt during ventricular systole. At the same time stenosis should not be produced. This eventuality had been foreseen in our previous

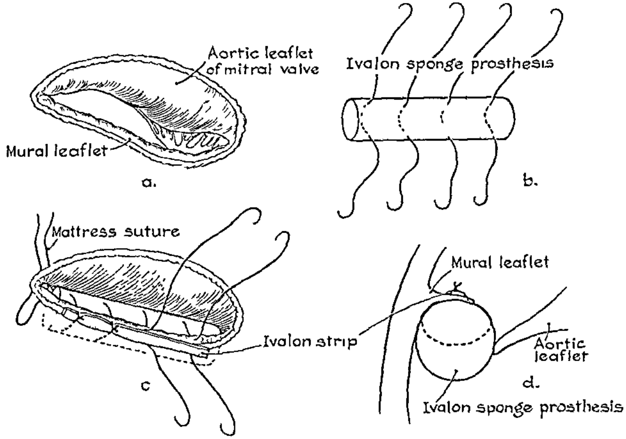

Fig. 11.—Correction of mitral regurgitation by direct vision insertion of a prosthetic leaflet.

a, This procedure is used alone or with annuloplasty where loss of valve substance is prominent. In this case the entire mural (posterior) leaflet is scarred and retracted. The aortic (anterior) leaflet is of good quality. The annulus fibrosus is of normal circumference.

b, Silk stitches (No. 2) with swedged needles are passed through an appropriately sized cylinder of compressed (3:1 or 4:1) polyvinyl sponge near its equator.

c, The mitral valve is viewed from above through the atriotomy incision and these needles are introduced from below upward. For each stitch, one needle is brought through the annulus fibrosus and its mate slightly adjacent as a verticle mattress stitch. Each of these stitches is then securely tied over a strip of polyvinyl sponge.

d, Portrays how this prosthesis restores competency without producing stenosis by providing a firm buttress against which the aortic leaflet may close.

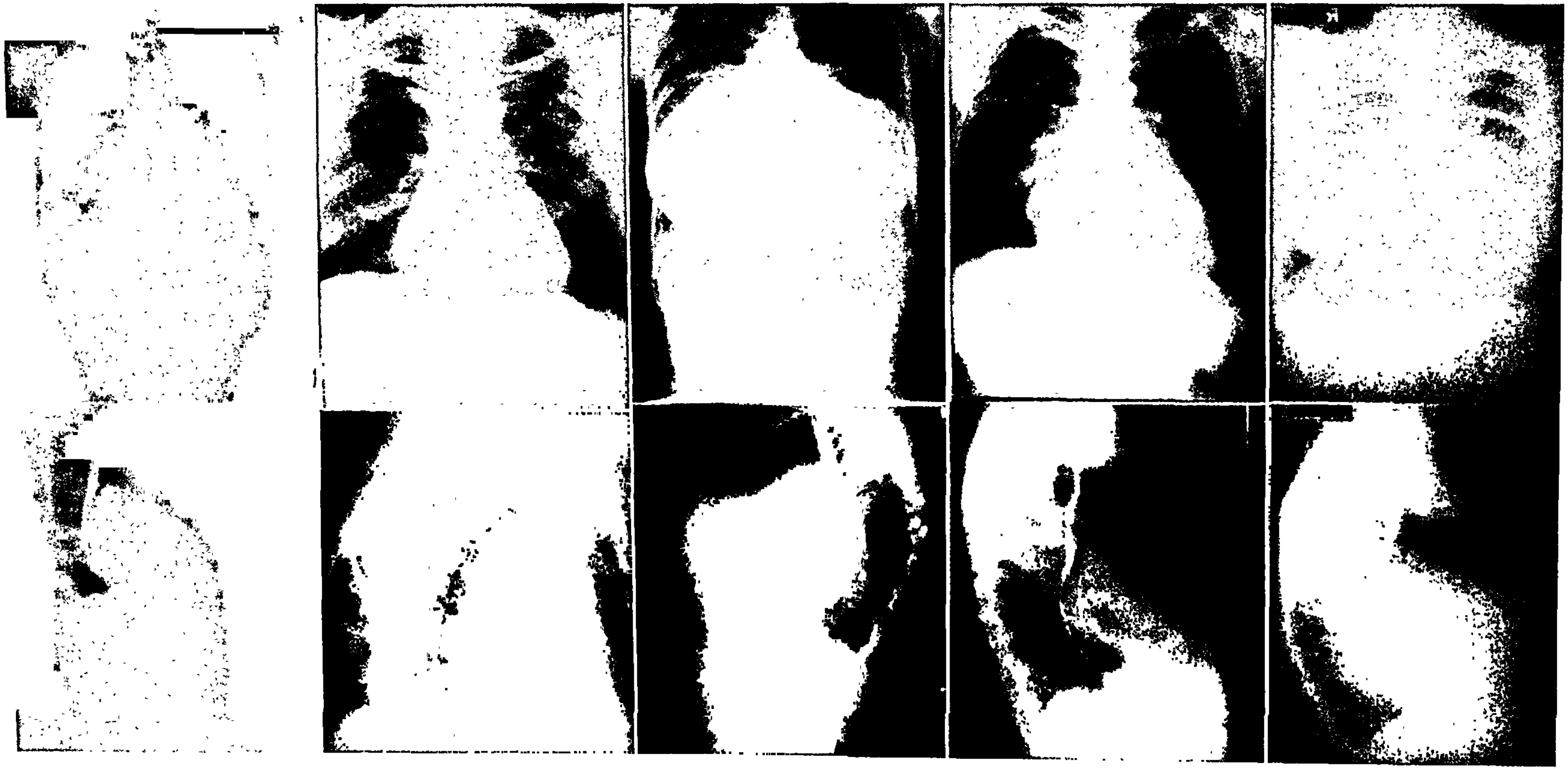

Fig. 12.—The preoperative frontal and lateral roentgenograms of the 5 patients undergoing correction of mitral regurgitation. (Cases 1-5 are arranged left to right.) The advanced states of cardiac disability are evident. There has been no operative or late mortality.

studies on autopsy specimens. Therefore various sized, compressed, polyvinyl sponge cylinders had been prepared in readiness for insertion as described below (Case 5 and Fig. 11).

Preparation and insertion of polyvinyl cylinders for mitral leaflet replacement: Earlier experimental studies[21] indicated that Ivalon cylinders, compressed 2 to 1 or more, changed very little in size over time after being inserted into the dog heart. Moreover, this material prepared in this fashion was well tolerated in the left side of the heart without creating emboli. These compressed Ivalon surfaces became completely covered by a smooth layer of fibrin in 6 weeks.

Cylindrical metal molds of several diameters, ranging from 1.2 cm. (½ inch) to 1.9 cm. (¾ inch), were fabricated and an assortment of polyvinyl cylinders were prepared at 3 to 1 and 4 to 1 compressions. They are sterilized by boiling the mold and the contained sponge for 30 minutes just before use.

In Case 5, a cylinder, 4 cm. in length and 15 mm. in diameter, compressed 4 to 1 was used. Four No. 2 silk stitches attached to a double-armed swedged-on needle were each passed through the cylinder near but slightly to one side of its equator as shown in Fig. 11, *b*.

Then, with care to keep these preplaced stitches oriented, the needles were passed from below upward coming directly through the annulus of the contracted and scarred mural leaflet. The opposite needle of each stitch was then placed similarly from below upward, 3 to 4 mm. medial (toward the free edge of the mural leaflet) as a vertical mattress stitch as portrayed (Fig. 11, *c*). Then, each stitch was tied over a narrow strip of compressed Ivalon.

This cylindrical prosthesis replaced the lost substance normally provided by the mural (posterior) mitral leaflet and became a firm buttress against which the aortic (anterior) mitral leaflet closed (Fig. 11, *d*). A gratifying feature observed in the previously mentioned animal studies was the absence of any tendency for the free leaflet to form adhesions to this Ivalon cylinder (see Fig. 1, reference No. 21).

Report of Cases.—

During the past 14 months at the University of Minnesota Hospitals, 5 patients, all far advanced in their clinical stage of the disease, have undergone direct vision reconstruction of the mitral valve for pure mitral insufficiency.[35-37] In 4 of these patients, we have been favorably surprised to find valve leaflets that were remarkably good, in fact they were virtually normal in their consistency. The regurgitant flow seemed to be due primarily to dilation of the annulus fibrosus about the mitral valve. In these patients, surgical repair was made by annuloplasty. In the fifth patient the annulus was of normal circumference, and the regurgitation was due to a severe loss of substance involving the entire mural (posterior) leaflet of the mitral valve. Repair in this patient consisted of insertion of a prosthesis of compressed polyvinyl plastic sponge (Ivalon) together with one annuloplasty stitch. Details concerning these 5 patients are presented below and summarized in Table III. The preoperative chest roentgenograms for these patients are reproduced in Fig. 12.

TABLE III. SURGICAL REPAIR OF PURE MITRAL INSUFFICIENCY UNDER DIRECT VISION UTILIZING THE PUMP-OXYGENATOR

CASE AND AGE	WEIGHT (KG.)	ETIOLOGY OF LESION	CORRECTIVE PROCEDURE	LEFT ATRIAL PRESSURES (MM. Hg)		DURATION OF BYPASS	FLOW RATE (C.C./MIN.)	PLASMA HEMO-GLOB. (MG. %)	ARTERIAL pH		CLINICAL IMPROVE-MENT
				PREOP.	POSTOP.				PREOP.	POSTOP.	
Case 1 D. D., 15	28	Fibroelastosis?	Annuloplasty	43/26	18/10	23′ 35″	1 200	15	7.62	7.63	Good
Case 2 W. D., 43	75	Rheumatic	Annuloplasty	48/20	30/20	25′ 38″	2,600	62	7.50	7.49	Excellent
Case 3 N. B., 19	45	Rheumatic	Annuloplasty	30/15	20/12	28′ 50″	2,300	61	7.44	7.51	Excellent
Case 4 W. O., 39	56	Rheumatic	Annuloplasty	50/20	17/5	22′ 22″	3,000	59	7.41	7.43	Excellent
Case 5 R. G., 43	59	Rheumatic	Insertion of valvular prosthesis together with annuloplasty	—	—	35′ 21″	3,500	50	7.43	7.39	Excellent

Case 1.—D. D., a boy of 15, had had a murmur detected at 9 months of age. When he was 11 years old, a coarctation of the aorta had been resected, and the presence of mitral insufficiency was confirmed by exploratory cardiotomy. At 12, he developed irreversible atrial fibrillation and progressive cardiac deterioration. At the time of surgery on Aug. 29, 1956, he was in American Heart Class IV E, being confined to bed with intractable right and left heart decompensation. Despite digitalization to the toxic level and complete bed rest, a persistent tachycardia was present. He was considered terminal by his cardiologist,* parents, and ourselves. He had a Grade 4 systolic murmur and thrill at the apex. At operation both mitral valve leaflets were slightly thickened but otherwise appeared virtually normal both as to consistency and leaflet area. The annulus was greatly dilated and the valve function revealed marked regurgitation due to this. Operative repair consisted of placing five No. 2 silk mattress sutures in the posterior aspect of the annulus and tying them down over pillows of compressed Ivalon sponge. This suturing completely obliterated the regurgitant jet, and there was no thrill in the atrium at completion of the perfusion run. The left atrial pressure was reduced from 43/26 before to 18/10 mm. Hg after the reparative procedure (Fig. 13). In the immediate postoperative period his improvement was rapid. A most gratifying objective response was a 12-pound weight loss during this interval from diuresis of the chronic edema fluid in his tissues. Fourteen months after the operation he is doing well and has been able to return to school. Digitalis is still required to control the atrial rate but this remains at 75 to 90 when he is ambulatory. The etiology of the mitral insufficiency in this patient remains uncertain although endocardial fibroelastosis has been suspected.

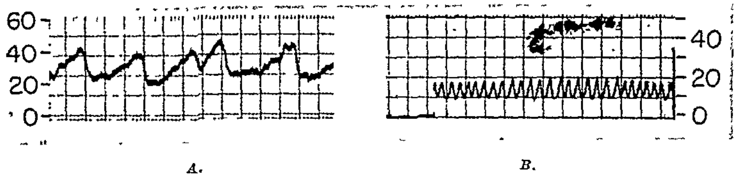

Fig. 13.—Mitral regurgitation, left atrial pressures (D. D., aged 15 years). The correction of valvular incompetence as manifested by the immediate reduction in left atrial pressures in our first patient undergoing annuloplasty in 1956 (Case 1, Table III)

A, Before correction, 42/22 mm. Hg. *B*, After correction, 18/10 mm. Hg.

Case 2.—W. D., a 43-year-old man, had had no history of rheumatic disease, but atrial fibrillation was noted on the electrocariogram in 1948. Since then he had experienced progressive cardiac failure, poorly controlled by digitoxin. He had a laparotomy for mesenteric embolus in 1953. A cerebral embolism 6 months before this present admission resulted in a left hemiparesis from which he was gradually recovering. At the time of this operation on his mitral valve, Dec. 28, 1956, he was in American Heart Class IV D and had Grade 3 systolic and diastolic murmurs at the apex. At operation the mitral valve was heavily calcified anteriorly. Stenosis was present by valve fusion at the anterior commissure. A very large regurgitant jet existed posteriorly. The anterior commissure was incised for 3 cm. and two No. 2 silk mattress stitches were tied over Ivalon to delimit the posterior extent of the annulus. No atrial or ventricular thrill could be felt after completing the perfusion. The atrial pressure was reduced from 48/20 to 30/20 mm. Hg after the annuloplasty. The patient, who had not worked during the 7 years before surgery, now has returned to a full-time position as an x-ray technician.

Case 3.—N. B., a 19-year-old girl, had had rheumatic fever at 14 years of age followed by repeated hospitalizations for episodes of heart failure. Despite digitalis therapy and rigid salt restriction this young woman had chronic heart failure leading to jaundice from cardiac cirrhosis. At the time of surgery she was in American Heart Class IV D with a

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*Dr. Herman K. Hellerstein, Cleveland, Ohio, to whom we are indebted for insisting that we operate upon this first patient.

Grade 4 systolic murmur and thrill at the apex. At the operation, the left atrium was fantastically enlarged (Fig. 12) and the mitral annulus was proportionately dilated. On the other hand the leaflets were not calcified but slightly thickened. The most evident locus of insufficiency was posterior but a significant reflux existed at the anterior pole also. An annuloplasty was created by placing No. 2 silk mattress sutures in the posterior aspect of the annulus and one mattress suture anteriorly. After these had been tied down over Ivalon, the valve was much more competent, and the atrial pressure was reduced from 30/15 to 20/12 mm. Hg. Postoperatively, only a soft Grade 1 systolic murmur was present. This follow-up has been short, but the patient is improved dramatically. Her salt restriction has been eliminated and she is on a regular diet now for the first time in years. Digitalis has been continued to control the heart rate. Evaluation of the result in this case is best summarized in her own words, "A New Life."

CASE 4.—W. O., a 39-year-old man, had had rheumatic heart disease at 21 years of age. Heart failure developed one year before this operation. He was well compensated on digitoxin, but he was unable to work as a farmer because of lack of physical stamina. Attacks of paroxysmal nocturnal dyspnea were frequent during the year just prior to this admission. At the time of surgery he was in American Heart Class III D and had a Grade 3 systolic murmur at the apex in addition to a Grade 2 diastolic murmur at the apex and atrial fibrillation. At operation, the mitral valve was widely regurgitant with slight stenosis at the anterior commissure. This stenotic anterior commissure was incised 1 cm. and the regurgitant channel through the posterior commissure was obliterated by two No. 2 silk sutures tied over Ivalon which narrowed the annular ring. The left atrial pressure was reduced from 50/26 to 17/5 mm. Hg and, postoperatively, only by means of phonocardiography was it possibly to detect any systolic murmur. His follow-up has been short too, but the patient is now on a regular diet and not taking digitalis.

CASE 5.—R. G., a 43-year-old woman, had had rheumatic fever at age 12 with the onset of atrial fibrillation at age 23 and then episodes of cardiac decompensation recurrent until the time of this surgery. A therapeutic abortion had been performed when the patient was 25 because of severe decompensation in the fourth month of gestation. Paroxysmal nocturnal dyspnea, ascites, ankle edema, and frequent episodes of acute pulmonary edema were prominent in her history. She had been on digitalis and a low salt diet continuously for 20 years. At the time of her admission she was in American Heart Class III D and had a Grade 3 systolic murmur at the apex. She went into shock as soon as she was anesthetized. Her heart action was very feeble and the systemic arterial blood pressure had dropped to 60 mm. Hg and hovered at this critical level during the chest opening period. In the race to perform the cannulations and put the patient on the pump-oxygenator before cardiac action failed entirely, it was deemed unwise to take time for measuring the left atrial pressures. Once the patient was linked to the pump-oxygenator her status immediately improved, and the operation could thereafter proceed at a more leisurely pace.

Upon opening the left atrium, a slight stenosis at the anterior commissure and severe posterior regurgitation were evident. Since the mural cusp of the mitral valve was virtually absent, a cylinder of compressed Ivalon sponge (4 cm. in length and 15 mm. in diameter) was placed horizontally underneath this structure and sutured in place as described (Fig. 11). One annuloplasty mattress stitch of No. 2 silk tied down over the Ivalon shortened the posterior annular segment. The regurgitant jet was almost completely abolished by these procedures. The patient's heart took over immediately and established a normal blood pressure at the conclusion of the perfusion. Postoperatively only a Grade 1 systolic murmur was audible at the apex. To date, this patient, like the others, has been impressively improved. For the first time in 20 years she is able to eat a regular salt content diet. It is quite obvious to us from her precarious condition just after being anesthetized and during thoracotomy that she could not have survived any operative procedure which did not immediately and substantially improve her mitral function.

Discussion —

As a group, these patients undergoing correction of mitral insufficiency have been among the sickest patients preoperatively upon whom the authors have undertaken cardiac surgery Thus, while the series operated upon remains small, the lack of operative or late mortality and the gratifying improvement, self evident in each instance, clearly indicates the value of the described reparative procedures carried out upon the incompetent mitral valve under direct vision

As already mentioned in reference to surgery upon the aortic valve, there will likely be encountered occasional patients in whom the valve leaflets have undergone virtually total destruction and complete valve replacement will be the only acceptable surgical treatment This successful replacement of much of the mural (posterior) mitral leaflet, the one most frequently damaged secondary to valvulitis, represents an important advance in that regard

As the principles and our knowledge of reconstructive procedures increase, it is evident that this ultimate approach of complete mitral or aortic valve replacement may be necessary much less frequently than we once had thought From this admittedly limited experience, we now believe that the surgical treatment of the primarily insufficient mitral valve may well become a more truly corrective procedure than that of the blindly performed operation for mitral stenosis because of the much better quality of the leaflet tissue in most patients with the former condition

MITRAL STENOSIS

The individual with pure mitral stenosis in need of surgical relief has a good chance of being benefited by a carefully performed valvotomy by the indirect or blind techniques classically described by Bailey, Harken, and others The principle problems remaining in this field for cardiac surgeons are those found in the patients with complicating factors which have been found to increase mortality and morbidity while lessening the palliation achieved While some surgeons have sought to overcome these problems by describing various technical and instrumental variations, the obvious solution seems to us to be a direct vision approach for these patients

Present Indications for An Open Operation Utilizing the Pump Oxygenator for Mitral Stenosis —

1 The considerably higher operative risk recorded for closed mitral commissurotomy performed in patients with clinically far advanced states of disability (Class IV)

2 A history of embolization prior to surgery or known or suspected large atrial clots or tumors

These two indications will also encompass many patients with unsuspected left atrial myxomas

3 Patients with apparent restenosis after a seemingly adequate valvotomy

4 Presence of or any suspicion of mitral regurgitation

TABLE IV. SURGICAL REPAIR OF MITRAL STENOSIS UNDER DIRECT VISION UTILIZING A PUMP-OXYGENATOR

CASE AND AGE (YR.)	SEX	WEIGHT (KG.)	LESION	ETIOLOGY	DATE OF OPERATION	PROCEDURE	DURATION BYPASS (MIN.)	CLINICAL RESULT
Case 1 3	M	13	Mitral stenosis; ventricular septal defect	Congenital	3/29/56	Rt. ventriculotomy; closure V.S.D.; left atriotomy; mitral valvotomy	38	Excellent
Case 2 52	F	40	Mitral stenosis; aortic regurg. and stenosis	Rheumatic	5/23/56	L. atriotomy; mitral valvotomy; aortotomy; aortic valvuloplasty*	18	Excellent
Case 3 3	M	9	Mitral stenosis; ventricular septal defect	Congenital	6/4/56	Rt. ventriculotomy; closure V.S.D.; left atriotomy; mitral valvotomy	14	Excellent
Case 4 41	F	56	Mitral stenosis†	Rheumatic	1/16/57	Left atriotomy; mitral valvotomy	19	Good
Case 5 13	F	34	Mitral stenosis;‡ aortic regurg.; tricuspid regurg.	Rheumatic	2/27/57	Left atriotomy; mitral valvotomy	29	Died

*Retroperfusion of coronary sinus for 13 minutes. (This is same patient as Case 3, Table I.)

†Mitral regurgitation was suspected preoperatively, but only pure stenosis was found at operation.

‡Had previous mitral valvotomy with no improvement.

5. Patients with multivalvular disease or other intracardiac lesions requiring an open procedure.

Direct vision surgery upon the mitral valve has been a very satisfactory procedure and there is little doubt that the indications for an open procedure are likely to be progressively widened in the future as experience with this procedure grows in various centers.

Surgical Technique for Open Mitral Valvotomy.—

The thoracotomy incision is the same as that already described for mitral regurgitation and aortic valve disease; namely, a right posterolateral thoracotomy. This same incision, of course, provides excellent exposure to the tricuspid valve should significant disease be found there. The left atriotomy has been performed anterior to the insertion of the pulmonary veins.

Rather unexpectedly one of the unanticipated problems of direct vision surgery for mitral stenosis has been the finding that in the congenital lesions as well as in some of the acquired stenoses of the mitral valve the line of fusion of the leaflets may be so perfect that no visual trace of the commissures remains. In such cases the line of attachment of the chordae tendineae has proved to be a valuable guide and landmark for making the proper division. Usually we have utilized the ordinary surgical scissors and scalpel for these direct vision commissurotomies. The valve leaflets, chordae tendineae, and papillary muscles are carefully inspected visually. Then a finger is inserted underneath the leaflets between the chordae tendineae of the respective leaflets and if the line of fusion of the leaflets is not apparent the incision is made upon the finger.

Results.—

Five patients have had direct vision mitral commissurotomies for mitral stenosis through a left atriotomy incision during total cardiopulmonary bypass utilizing the pump-oxygenator. We have had one operative death, no late deaths, and in the other 4 the results have been successful. All 5 of these patients had other complicating lesions as indicated above as a basis for their selection.

Relevant data upon these patients are listed in Table IV.

In both of the patients with congenital mitral stenosis associated with a congenital ventricular septal defect we have been alerted by our pediatric cardiologists* to the possibility of a coexisting mitral stenosis because of certain unusual features in their work-up. These were varying combinations of: an unusually loud diastolic murmur of mitral quality heard best at the apex, unusually marked left atrial enlargement noted at cardiac fluoroscopy, notching of the P waves in the electrocardiogram and greater than anticipated pulmonary wedge pressures measured at cardiac catheterization. None of these findings is diagnostic in itself, but are sufficient to arouse a suspicion of this associated defect. At surgery both patients had prominent diastolic thrills present in the left ventricle. In these patients we opened the r [illegible] and repaired the ventricular septal defect first and then restored the circulation. The left ventricular diastolic thrill persisted in both and so the pump-oxygenator was turned on again and the left atrium opened for the repair. This sequence rules out the possibility of opening the left atrium needlessly for a relative mitral stenosis which can occasionally occur in a patient with an unusually large pulmonary blood flow.

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*Drs. Ray Anderson and Paul Adams.

The one death was in a 13-year-old girl with a definite history of rheumatic fever beginning at the age of 5 years. In March of 1956, during a classic mitral commissurotomy at another medical center, the heart arrested and after some difficulties the beat was restored. Convalescence was incomplete and chronic right and left decompensation persisted despite limiting her activity, hospitalization, and the other usual medical methods. Right heart catheterization disclosed severe pulmonary hypertension (85/35 mm. Hg) and no shunt. Left heart catheterization disclosed a left atrial-left ventricular diastolic gradient of 26 mm. Hg with no evidence of mitral incompetence but with the typical findings and murmur of aortic insufficiency. She also had tricuspid regurgitation. At operation, the mitral valve was severely stenotic and calcified and although both commissures were readily opened to the annulus relatively little flexibility of the leaflets remained. Intermittent aortic occlusion was necessary to control blood loss from the aorta during the left atriotomy. No attempt was made to attack the tricuspid or aortic regurgitation. Although the operative procedure was uneventful and her immediate postoperative status seemed good, cardiac arrest occurred without warning later on the day of operation. In such a patient now we would advocate the surgical treatment of the other valvular lesions at the same time.

SUMMARY AND CONCLUSIONS

1. Direct vision reparative procedures making use of total cardiopulmonary bypass with a pump-oxygenator have been described for aortic stenosis, aortic regurgitation, mitral stenosis, and mitral regurgitation.

2. New operative techniques, made possible by the increased accuracy and scope afforded to the surgeon by direct vision, have been devised for the correction of aortic regurgitation and mitral regurgitation. The results have been particularly successful for those patients afflicted with mitral regurgitation.

3. Currently all patients with aortic stenosis, aortic regurgitation, or mitral regurgitation and in need of surgical treatment are best managed by an open bypass operation. Patients with pure mitral stenosis and free of complicating factors which are known to increase morbidity and mortality are treated by the closed mitral valvotomy. The indications for an open operation for mitral stenosis are the presence of one or more of these above-mentioned additional complicating factors. For the future, it is likely that these indications for a direct vision procedure for mitral stenosis will be progressively broadened.

4. The unilateral right posterolateral thoracotomy through the bed of the fourth or fifth rib offers excellent access to the aortic or mitral valve areas

and the good exposure afforded by use of this incision has been an important factor in the successes obtained. Occasional patients with other congenital lesions in the left chest may require a bilateral anterior thoracotomy.

5. Retrograde perfusion of the coronary sinus in combination with a pump-oxygenator has been used in 8 patients for a direct vision aortic valvuloplasty.

6. Five of the 8 patients with aortic valvular disease survived the operative procedure and attained excellent clinical improvement following direct vision valvuloplasty. Two of the 3 deaths occurred in infants. The other failure was due to the production of aortic insufficiency in a patient with a severe distortion and calcification of the valve due to acquired disease. Now, we believe that it is better for patients with such rigid and distorted valves to relieve stenosis by creation of a bicuspid aperture. In the other acquired lesions with less advanced destruction and a greater degree of flexibility remaining, restoration of the triangular orifice still is sought for.

7. In all of these operations the retroperfusion of the coronary sinus appeared to protect the myocardium well against anoxia. There were no instances of cardiac arrhythmia even though a significant percentage of the patients had far advanced left ventricular disease.

8. Acetylcholine has also been used in combination with the retroperfusion in 2 patients. It was injected into the retrograde catheter and provided a dry still field while the aortic valvuloplasty was being performed. Both patients had an excellent clinical result.

9. Total cardiac ischemia of brief duration resulted in extensive myocardial necrosis in a patient with acquired aortic stenosis, and this approach is not recommended for aortic valve surgery.

10. Evidence is advanced to support the belief that total cardiac ischemia, even with the additional protection offered by potassium citrate asystole, is also a dangerous practice in hearts afflicted with the advanced states of aortic valve disease due to an inability to restore a left ventricular beat in both of the only 2 patients in which it was utilized.

11. Acetylcholine asystole with total cardiopulmonary bypass has been utilized in 6 patients with aortic valvular disease, and has proved of value when the arrest interval was brief. However, a significantly greater incidence of arrhythmias occurred than when retroperfusion was utilized. One operative death in this group occurred in a patient with acquired disease in whom a more prolonged procedure was required.

12. Current practice has been to use the acetylcholine arrest in patients with congenital lesions where the operative repairs require less time and the myocardiums generally have a greater reserve. Retrograde perfusion of the coronary sinus, together with retrograde acetylcholine cardioplegia if needed to facilitate exposure, is the recommended procedure for all patients with advanced degrees of acquired aortic valvular disease.

13. Although acetylcholine cardioplegia (without retroperfusion) appears to be the simpler technique for aortic valve exposure, retrograde perfusio s

undoubtedly the safer method when there is marked myocardial hypertrophy and a longer period is contemplated for the valvuloplasty.

14. The concept of correcting visually either surgically created or primary aortic regurgitation by converting the valve leaflets (by suture) to a bicuspid orifice and prosthetic replacement to make up for any shrinkage or loss of tissue substance is offered. Utilizing this method, the first patient to have a successful complete correction of primary aortic insufficiency is reported.

15. Two new operative techniques have been described for the correction of mitral insufficiency without the production of any stenosis. One procedure, termed annuloplasty, consists in part of selective suturing of the annulus fibrosus under direct vision to reduce its circumference commensurate with the area of valvular tissue present. The other procedure for use in patients with substantial loss of leaflet substance is the insertion of a prosthesis.

Five patients have been treated by these techniques to date. Before surgery all had been seriously incapacitated by severe mitral regurgitation. There has been no operative or late mortality. All are alive and significantly, even dramatically, improved. Moreover, the first patient, whose progress has been followed for more than a year, has not only sustained his initial gains but has continued to improve. Thus, although the series is small, the results indicate that surgical correction of mitral insufficiency is feasible, effective, and lifesaving.

16. Five patients with pure mitral stenosis of acquired or congenital etiology together with other complicating factors or lesions have been treated by a direct vision approach. There was one operative death in this group. The 4 survivors have been benefited significantly.

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DISCUSSION

(PAPERS BY SWAN [PAGE 139], LILLEHEI [PAGE 154] AND THEIR ASSOCIATES)

DR. PAUL W. HERRON, Seattle, Wash.—At the University of Washington to date we have operated on 5 patients with mitral lesions using the pump-oxygenator. Three of these had pure mitral insufficiency and all survived the immediate procedure. Two of them had very excellent results. I would like to show two slides.

(slide) This shows the left atrial pressure taken before and after repair of the insufficient valve, and shows a marked reduction in left atrial pressure after correction.

(slide) This is a chest film taken before and 2 weeks after surgery. The change in heart size is apparent.

(movie) I would like to show an excerpt of a movie taken during repair on one of these patients which indicates a possible new approach to the left atrium. It utilizes a right thoracotomy, as advocated by Dr. Bailey, but approaches the left atrium posterior to the pulmonary hilum rather than anterior. We feel this approach may make the mitral valve even more accessible than Dr. Bailey's approach. As yet we have not used this method but will at the next opportunity. We offer this approach for your consideration.

DR. GEORGE H. A. CLOWES, Cleveland, Ohio.—I agree perfectly with the authors that the way to approach this problem is to do it by open surgery. My associate, Dr. Neville, and I have operated upon 2 adult patients with mitral regurgitation using the membrane oxygenator. In both instances, perfusion was quite successful. The first patient has made a very satisfactory recovery. We opened the left auricle and by dividing the commissures and four chordae tendineae under direct vision we got the result we wanted without putting any stitches into the valve itself.

The next case—and I think this is more important—was a failure. The perfusion went perfectly well, the patient made a reasonably good recovery, but died very suddenly with acute pulmonary edema the night after the operation. This patient had been previously operated on and a commissurotomy was done. She had had two diagonal cuts into the major leaflet in the wrong place that had allowed the major leaflet to flap and had created a regurgitation. We had located these things under direct vision, at operation put the commissures in the proper place and then put a stitch into each of the two cuts. When we left the operating room, there was no regurgitation and we thought we had an excellent result. I regret to say that the next morning at autopsy, one of the stitches had cut out and she had a wide-open regurgitation again. It appears that mitral valve lesions will not hold stitches under these circumstances.

The other thing is with regard to Dr. Lillehei's remarks on the necessity for perfusion of the heart during the period of occlusion when one has the aorta open. In a series of dogs some years ago which was reported at a Forum in Atlantic City, we never failed to defibrillate and get a recovery in any period under 20 minutes when the animal was not having the coronaries perfused. On the other hand, if we went along for 30 minutes without any circulation in the heart, the great majority of these animals died of cardiac failure later if they could be defibrillated at all.

DR. DENTON A. COOLEY, Houston, Texas.—The presentation by Dr. Lillehei is most impressive and deserves high praise from all of us. At Baylor University we have used cardiopulmonary bypass in 155 patients with a variety of cardiac and aortic lesions, and I am convinced that this method offers optimum conditions for open cardiac procedures At present, I am not convinced, however, that retrograde coronary perfusion is necessary in exposure of the aortic valve for operation We have operated upon patients with aortico pulmonary septal defects, with aneurysms of the ascending aorta, as well as 9 with aortic stenosis, without retrograde coronary perfusion and have noted no ill effects On the other hand, in the first patient with calcific aortic stenosis in whom retrograde perfusion was used, ventricular fibrillation occurred immediately after starting the perfusion and cardiac resuscitation was unsuccessful I believe that exposure of the aortic valve through an ascending aortotomy can be achieved safely without coronary sinus perfusion and valvulotomy accomplished within 5 minutes For this period of time, the ventricular myocardium will usually continue to contract regularly with a sinus rhythm After operation we have detected no evidence of myocardial damage caused by this brief period of ischemia I shall be interested to compare our results with Dr Lillehei's after we have both had more experience

DR CHAS P BAILEY, Philadelphia, Pa —[The corrected discussion by Dr Bailey has not been returned by the discusser —EDITOR]

DR C WALTON LILLEHEI, Minneapolis, Minn —In regard to Dr Herron's suggestion of entering the left atrium behind the right pulmonary hilus I believe it is relatively unimportant whether the surgeon chooses to make his atriotomy incision there or anterior to the hilus The mitral valve exposure is excellent by either route The technical consideration that has seemed important to success has been the choice of the right posterolateral thoracotomy incision to approach the left atrium

With regard to Dr Cooley's and Dr Clowes's comments about stitches in the mitral valve, I emphasized that the annuloplasty stitches are not placed in the valve leaflet tissue at all, and should not be in my opinion, but rather are placed in the annulus fibrosus of this valve from above through the left atriotomy approach

Another point that I sought to emphasize was the fact that there is a remarkable difference in the tolerance to elective arrest techniques especially those involving the profound relaxation induced by potassium citrate between the human heart with advanced degrees of aortic valve disease when compared to hearts from patients with many types of congenital defects Suffice it to say that these hearts with massive left ventricular hypertrophy, often with superimposed scattered areas of fibrosis as well, due to actual or relative coronary insufficiency tolerate potassium asystole or any form of even brief hypoxia rather poorly Certain reasons for this observation have been suggested in the body of our paper Moreover, as our experience has increased, it has become increasingly evident that, save for exceptional cases of the congenital variety, it is not possible nor advisable to complete an adequate aortic valuloplasty within a few minutes

The surgical correction of pure mitral insufficiency carried out under direct vision has proved to be a very satisfactory surgical procedure even in some very far advanced patients, approaching or actually in the terminal phases of their disease The reasons for this are both anatomic (the frequent finding of valve leaflet tissue which is still soft and pliable even though inadequate in quantity) and physiologic (the fact that the hearts are immediately and significantly improved the moment this large leak is corrected which makes the procedure akin to closing an atrial septal defect or dividing a patent ductus arteriosus with a large shunt)

THE SURGICAL CORRECTION OF AORTIC INSUFFICIENCY BY CIRCUMCLUSION

WARREN J. TAYLOR, M.D. (BY INVITATION), WENDELL B. THROWER, M.D. (BY INVITATION), HARRISON BLACK, M.D., AND DWIGHT E. HARKEN, M.D.
BOSTON, MASS.

THE surgical treatment of aortic insufficiency has been disappointing. This discussion touches upon the natural evolution of aortic insufficiency, outlines a morphologic classification, discusses hemodynamic alterations, and reviews the surgical maneuvers evolved to date. A new operation for selected types of aortic insufficiency is presented.

NATURAL HISTORY OF THE DISEASE

Individuals with either rheumatic or syphilitic aortic insufficiency may remain asymptomatic for many years without treatment even while the left ventricle is enlarging. Many observers have emphasized that once symptoms appear, patients with aortic valvular disease deteriorate rapidly. The urgency of this axiom seems difficult to appreciate when dealing with the specific patient.

Segal, Harvey, and Hufnagel[21] have pointed out that aortic insufficiency predominates in males over females by three to one. In their experience, rheumatic origin has been more common than syphilitic. The interval between the onset of recognizable auscultatory rheumatic aortic insufficiency and the development of limiting symptoms may be as much as 10 years. The *initial* congestive failure is an ominous warning but generally responds to appropriate medical therapy. When angina pectoris appears it often heralds the terminal phase of the disease. Bland and Wheeler[3] have also documented the grave prognosis associated with this symptom, particularly at rest. Orthopnea, paroxysmal nocturnal dyspnea, and peripheral edema are late in the natural history of this disease and therefore of limited value in selecting the time for surgery. By contrast Bergeron, Ellis and co-workers[2] have found angina and syncope to be of lesser significance in aortic stenosis.

Syphilitic aortitis and associated insufficiency produce a clinical course comparable to rheumatic regurgitation, but the disintegration is much more rapid.[21] The study of Montgomery, Anderson, and Boone[17] indicates that the average survival time after the appearance of the murmur of aortic insufficiency due to syphilis is approximately 24 months. Aortic insufficiency with congestive failure or angina due to syphilis comes later in life than in rheumatic disease. If bacterial endocarditis is superimposed, the deterioration of the patient may be accelerated.

From the Department of Surgery of the Harvard Medical School at the Peter Bent Brigham Hospital and The Thoracic Service of the Mount Auburn Hospital.

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Read at the Thirty-seventh Annual Meeting of The American Association for Thoracic Surgery at Chicago, Ill., May 4-7, 1957.

DIAGNOSIS

Free aortic insufficiency of the type recognized by water hammer (Corrigan) pulse, diastolic murmur, and left ventricular dilatation is not commonly encountered as a pure lesion. More often there is an associated element of stenosis.

Gorlin and Case[8] have discussed the differentiation of stenosis from insufficiency. Throbbing, even pain over large arteries suggests insufficiency. Sweating, flushing, and episodes of tachycardia, as originally described by Lewis,[15] are more commonly associated with dominant aortic regurgitation. Sweating and flushing are due to peripheral vascular dilatation. Angina, syncope, dyspnea, and orthopnea appear late and are also characteristic of stenosis, so are not helpful in differentiation. DeMusset's sign (bobbing of the head) is usually associated with *severe* aortic insufficiency.

Certain aspects of the physical examination are helpful. Cardiac size, particularly early in the disease before the onset of failure, is often a clue to the dominant pathology. There is early enlargement of the left ventricle in aortic insufficiency. A comparable degree of enlargement appears late in aortic stenosis, and then with frank failure. Regurgitation produces increased activity of the apical impulse. Ancillary signs include bisferious (twin peaked) carotid pulse, Duroziez's sign, and capillary pulsations.

One of the most important differential observations may be made at fluoroscopy. In the left anterior oblique position, a rocking motion of the left ventricle and aortic arch is seen. This is due to the very large stroke output in systole and reflux in diastole. The extraordinary activity of the aortic arch described as "run off" is due not only to regurgitation but to accelerated forward flow into dilated peripheral vessels.

Aortic murmurs are notoriously misleading. Insufficiency without an accompanying diastolic murmur must be rare. Frequently there is an accompanying "diamond shaped" aortic systolic murmur transmitted upward to the neck and downward to the apex. The intensity of the murmurs does not quantitate the obstruction or reflux. A systolic murmur, due to turbulence over roughened cusps, may exist without organic stenosis, particularly in the presence of the increased stroke volume. Late in the course of the disease, murmur intensity may decrease due to myocardial failure rather than to any alteration in the valve. The Graham Steell murmur of pulmonic insufficiency may be readily differentiated if the other criteria of significant aortic insufficiency are sought.

Usually there is a wide pulse pressure, but its degree is not helpful in assessing the hemodynamic problem. Gorlin and Goodale[9] point out that the pulse pressure narrows as the systolic level falls and diastolic pressure rises "as the vasodilatation of insufficiency gives way to the low cardiac output and vasoconstriction of heart failure."

CLINICAL CLASSIFICATION

Although surgical experience with aortic regurgitation has been limited, a clinical classification of the disease is useful in evaluation and communication. The broad outline of the American Heart Association Classification is basic, but specificity for aortic insufficiency must be added just as in our clinical grouping for patients with mitral stenosis. The following classification *is suggested. It will be altered as experience dictates.*

Group I: Individuals with the murmur of aortic insufficiency who are asymptomatic and whose hearts are not enlarged more than 15 per cent over the predicted transverse diameter.

Group II: Individuals with the murmur and mild limitation due to dyspnea on exertion whose limitation is static and who do not require medical therapy.

Group III: Individuals who have the murmur of aortic insufficiency and significant symptoms, e.g., sweating, flushing, tachycardia, evidence of congestive failure, with or without angina pectoris. These symptoms are held static only by an adequate medical regimen.

Group IV: Individuals whose symptoms are refractory to a strict cardiac regimen.

Group V: Individuals who have refractory congestive changes in spite of a strict cardiac regimen.

MORPHOLOGIC TYPES

Rheumatic fever, syphilis, and bacterial endocarditis are the most common causes of aortic insufficiency.

Rheumatic incompetence of the aortic valve is conventionally ascribed to shortening of the leaflets. The cusps simply do not occlude the base of the aorta during diastole (Fig. 1). However, to this gross classical concept, the dimension of time must be added. When diseased aortic valves are observed on the MacMillan pulsator,* the leaflets not only fail to approximate due to lack of substance, but they move slowly, so that considerable reflux occurs before this inadequate closure (Fig. 1). These valves may or may not be calcific. In advanced stages, the dilatation or failure of the left ventricle frequently results in relative mitral incompetence with further burden. Insufficiency may be associated with minimal to dominant degrees of stenosis, with or without calcification.

Syphilitic aortitis with associated medial necrosis results in dilatation of the annulus and separation of the cusps at the commissures, rendering the valve incompetent. If the cusps themselves are involved, it is by fibrous thickening and fixation of the margins. There may also be cusp adherence to the walls of the sinuses of Valsalva (Fig. 1). This form is less common since the advent of penicillin.

A third important cause of aortic insufficiency may be from endocarditic cusp erosion (Fig. 1). Bacterial endocarditis is presumed to occur on previously diseased or congenitally abnormal valves (Fig. 1).

Other less common forms of aortic regurgitation include congenital malformations and traumatic rupture of cusps. Glendy and White[6] have stated that antecedent disease predisposes to cusp injury from blunt trauma.

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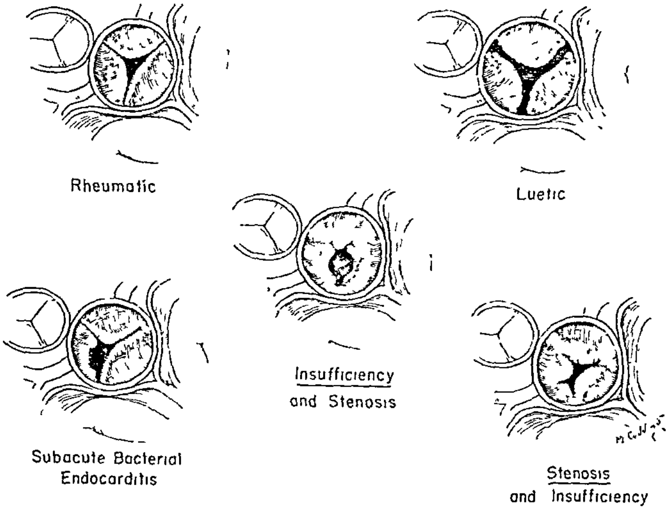
*Dr. Ian MacMillan and Dr. Stanley Sarnoff have kindly made a pulsator available for use at the Peter Bent Brigham Hospital.

HEMODYNAMICS

Left heart catheterization has added a valuable tool to clinical and investigative studies. Determination of the left ventricular pressure and pulse contour compared with brachial artery pressure, in the conscious patient, yields significant data about valvular lesions. The measurement of the aortic valve gradient is supplemented by injection of dye or radioactive material into the left auricle or ventricle. Sampling of brachial arterial blood gives a curve from which an estimate of insufficiency, between the points of injection and sampling, is derived. The regression formulas of Korner and Shillingford[14] have been modified and applied to left heart catheterization by Dexter and associates.[5]



Rheumatic

Luetic

Insufficiency
and Stenosis

Subacute Bacterial
Endocarditis

Stenosis
and Insufficiency

Fig 1.—Common morphologic patterns of aortic insufficiency.

This has been helpful in differentiating mitral stenosis and insufficiency.[4] It is to be hoped that a comparable degree of proficiency can be developed in the determination of aortic insufficiency. The problem of combined aortic and mitral insufficiency awaits solution.

Peripheral arterial pulse tracings are of little value in differential diagnosis due to the influence of myocardial failure and the degree of peripheral resistance.

PRINCIPLES OF SURGICAL CORRECTION

The burden of aortic insufficiency falls primarily on the left ventricle which may eventually fail. This failure is due to increased work in maintaining forward flow or to inadequate coronary blood flow at a lowered diastolic pressure.

A significant proportion of the added work appears to be relieved by the Hufnagel prosthetic ball valve placed in the descending aorta just beyond the left subclavian artery. Hufnagel and his colleagues[13] feel that this reduction of work may be of the order of 75 per cent. We have had discouraging experience with these valves. Placing the valves is not a problem; however, some patients have continued to have symptoms including increased angina. Sudden death has occurred. The diastolic pressure in the arms can be observed to fall after the insertion of the Hufnagel valve. Mean diastolic pressures have been shown to be lower by Rose, Hufnagel, Freis, Harvey, and Portenopen[19] and also by McKusick, Kahn, Brayshaw, and Humphries.[16] This may have bearing on the increase of angina following valve insertion, and the limited clinical success. It is appropriate to consider more physiologic placement of valves or repairing the incompetence below the level of the coronary ostia.

The same criticism directed at the placement of the Hufnagel valve far distal to the coronary ostia pertains to Murray's[18] use of an homologous aortic valve segment in the same position. The physiologic and hemodynamic disadvantages are compounded by the limitations of homografting itself.

Instead of replacing the valve, Sarnoff and Chase[20] have devised an L-shaped Lucite tube containing a ball valve that affords a new outflow tract at the cardiac apex. This inserts directly into the descending aorta. They have used this extensively in the laboratory. This or some similar principle may be useful at some time in the future.

Upon scrutiny of the morphology of aortic insufficiency and the principles involved in the local correction of regurgitation, at least four additional considerations are warranted.

First.—The first principle concerns incompetence associated with stenosis. At times the incompetence is not due to shortening of the leaflets but to inability of the leaflets to move into the closed position. Often these leaflets are held open in a pyramidal position by fusion at the commissures (Fig. 1). In this situation, fracture of the fused commissures, allowing more *complete* and more *rapid* closure, reduces the insufficiency and corrects the significant stenosis.

Second.—A second principle involves placing an obturator above the existing valve. This obturator, round or spherical in shape, is anchored by flexible moorings to the aortic wall; it moves away from the valve orifice in systole and back to obstruct reflux in diastole. Bailey[1] used such prostheses above combined stenotic and incompetent orifices.

Glenn and his associates[7] have employed a similar principle by placing material through mitral orifices. This substitute for loss of substance in cusp length and occlusive capacity also shortens the closure time of the leaflets. We have placed various substances through the aortic orifice in animals. The most promising would seem to be a braided Teflon cord, approximately 4 mm. in diameter (Fig. 2).

Third.—A third principle involves adding length and flexibility to the existing cusps. This may be accomplished by autografting or suturing material, such as Teflon, to the cusps.

Fourth.—The fourth principle pertains to the reduction of the size of the annulus of the aorta. In such a way, small leaflets may become adequate. Bailey's "sash"[1] employed this principle. This sash was placed so wide of the base of the aorta that it seemed unlikely to be effective. Similarly, commissural suture or neutral cusp plication has been considered. These have not, to our knowledge, had successful human application. Such plication, particularly total neutral cusp infolding, seems unreasonable when one considers the character of ventricular and aortic walls.

Finally, direct uniform reduction of the base at the annulus seems attractive because some pathologic processes producing incompetence are primarily due to dilatation, e.g., syphilitic lesions and diffuse myocardial dilatation. In other instances, the leaflets are shortened and remain flexible. These conditions are partially or completely defeated if there is extensive calcification of the leaflets or the annulus.

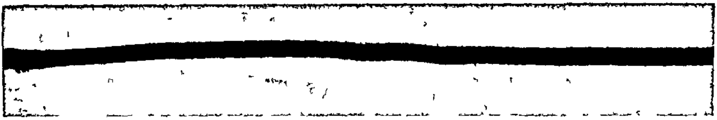


Fig. 2—Braided Teflon valve seat.

Ideally, circumclusion should be carried out through direct dissection beneath the coronary ostia rather than involving the widespread tissue compression mentioned in connection with the "sash" operation. Proper circumclusion involves placement of this constricting band below the widest girth of the truncated base of the aorta (Fig. 7). This constricting band must be low enough to reduce the area at the very base of the sinuses of Valsalva in order to afford maximum reduction of area to be occluded. The dissection is quite feasible when gently and delicately carried out.

TECHNIQUE FOR CIRCUMCLUSION

The patient is placed in a supine position with the right side elevated to an angle of 35 degrees. A right inframammary incision is made over the fourth interspace and extended transversely across the sternum to the left third interspace (Fig. 3). The right pleural cavity is entered through the fourth interspace. The sternum is transected obliquely connecting the fourth right with the third left interspace. The pericardium is opened obliquely to expose the great vessels and right ventricle. The pulmonary artery and ascending aorta are separated down to and through the conus ligament (Fig. 4). Anteriorly, dissection is carried toward the base of the aorta, reflecting the right ventricular myocardium and pericoronary fat. The right coronary artery is isolated (Fig. 4). The aorta is exposed 1 to 1.5 cm. below the coronary origin. Exposure of the posteromedially placed left coronary artery is more difficult, but it must be adequately visualized in order to place the circumcluding suture. The dissection is carried gently *below the widest diameter of the sinuses of Valsalva,*

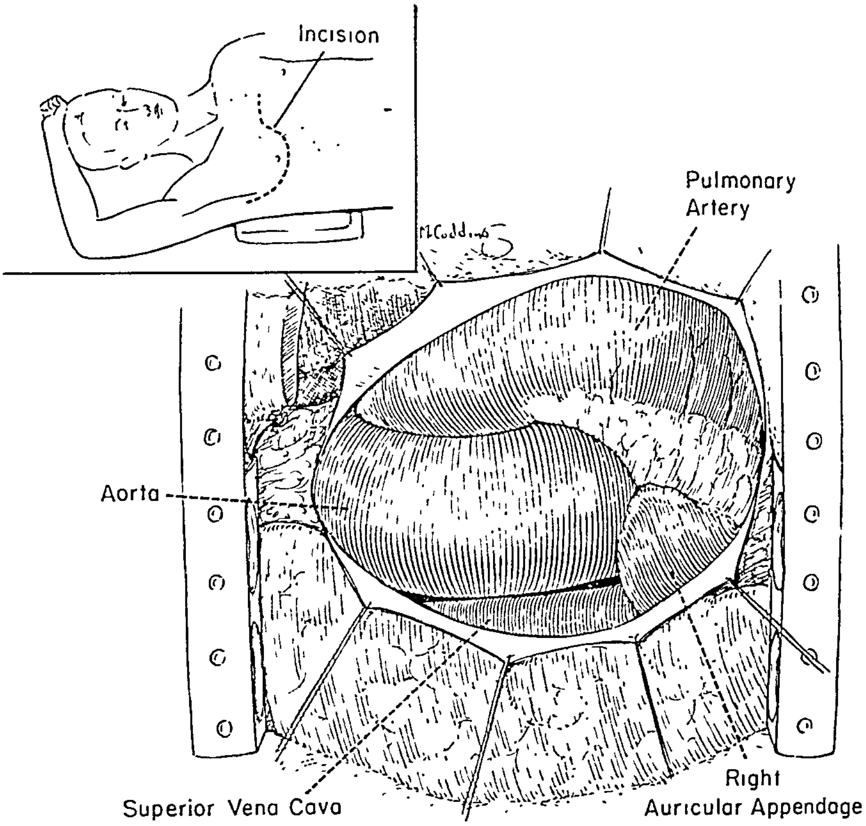


Fig. 3.—Right anterolateral incision and exposure.

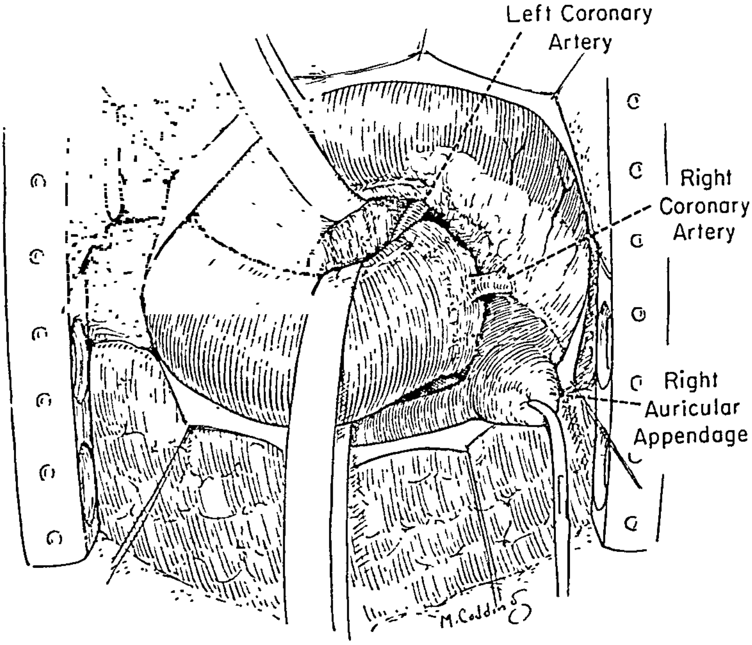


Fig. 4.—Details of coronary artery dissection. Division of conus ligament allows exposure of left coronary artery.

since the circumcluding suture must, on constriction, tend to migrate downward to reduce the aortic valve area, rather than upward and angulate the coronary arteries (Fig. 4). A double-tapered braided silk suture* is used. This suture tapers from No. 3-0 at the swedged malleable vascular needle to No. 5 in the middle and again reduces to No. 3-0 at the other needle (Fig. 5). The circumcluding suture is started lateral to the right coronary artery where a firm anchoring ligature is taken in the myocardium deep in the dissected groove at the base of the aorta. The suture is then passed beneath the right coronary artery (Fig. 6). Another anchoring stitch is placed under the divided conus ligament. The origin of the left coronary artery is underpassed and the left and right fibrous triangles, as described by Barry and Patten,[10] are incorporated in the suture line. The heavier tapered material is advanced so that the base of the aorta is circumscribed by the No. 5 segment. It is emphasized that the suture must be placed well below the origin of the coronary vessels and below the widest girth of the promontories of the sinuses of Valsalva, as described previously. In earlier dissection this was not adequately appreciated. Palpation of the base of the aorta by a finger within the right auricle facilitates placement of the stitch low in relation to the neutral (noncoronary) cusp (Fig. 6). This tends to ensure the low position of the circumclusion for more effective coaptation of cusps and protection of coronary arteries. Having thus placed the circumcluding suture (Fig. 7), it may be used alone or entwined with another No. 5 silk suture, or even cotton umbilical tape, to provide added bulk.

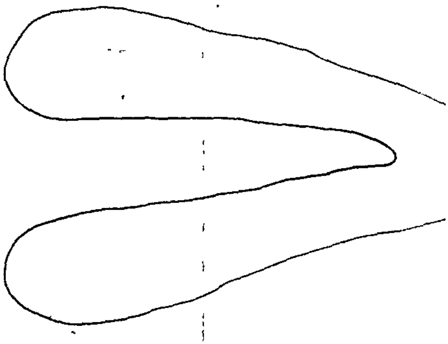


Fig. 5.—Double-tapered suture with malleable swedged vascular needles.

The degree of reduction of the aortic base is a delicate determination. As the constriction is increased, the cusps can be felt to close with a sharp impact in diastole. The anesthesiologist may detect an elevation of the diastolic pressure. In only one instance was intra-aortic digital control used as the suture

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*Ethicon, Inc., Somerville, New Jersey.

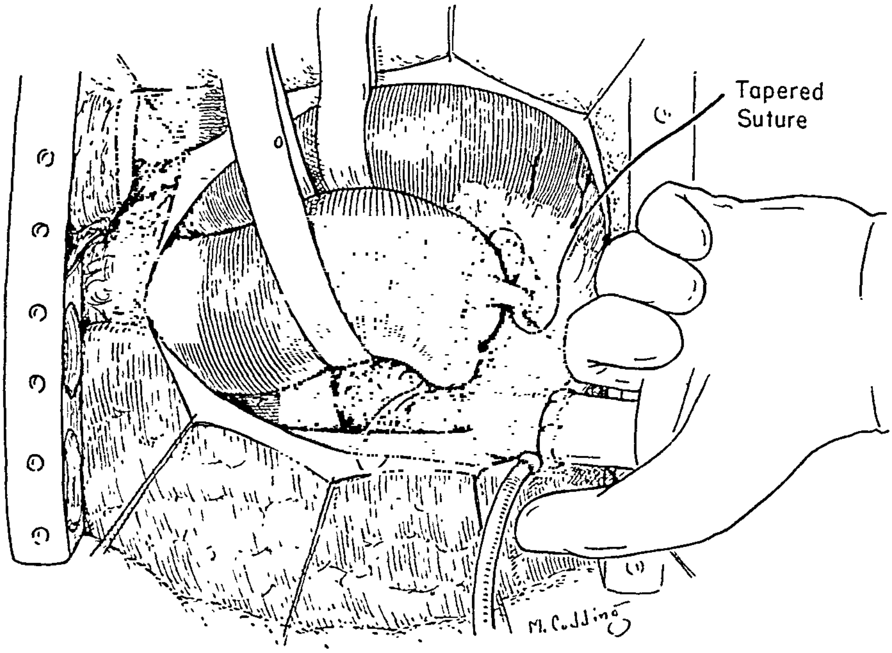

Fig. 6.—Double-tapered suture ligature passed under coronary arteries, anchored low in relation to neutral (noncoronary) cusp. Finger as guide is in right atrium.

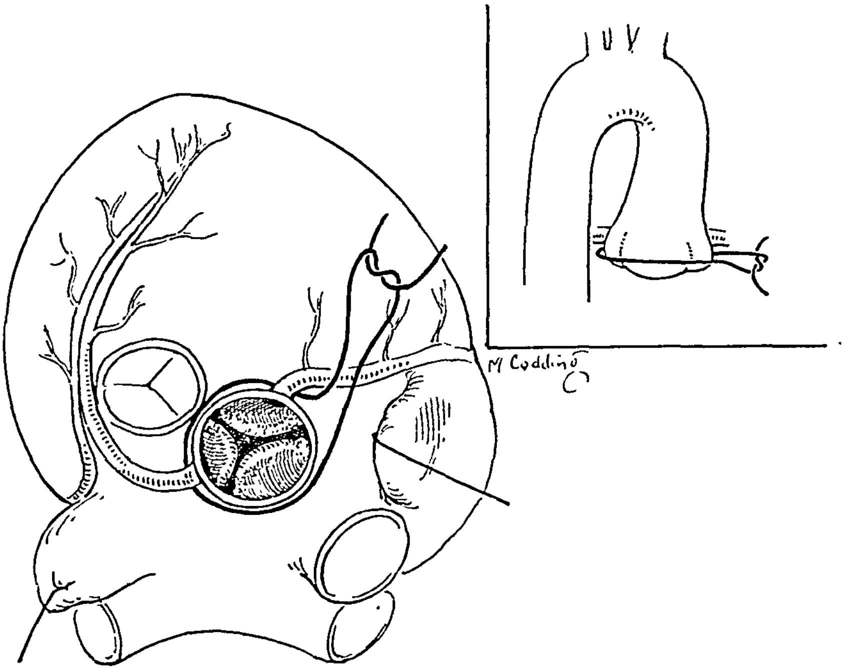

Fig. 7.—Location of circumcluding ligature.

was tied. This was accomplished by suturing an Ivalon operating tunnel to the ascending aorta, through which the surgeon palpated the valve as the tie was made. This seems unnecessarily complicated.

Operative Experience.—Eleven patients with pure aortic insufficiency have had the circumclusion operation. Five are living and improved. There were 4 operative deaths and 2 late deaths. All were males varying in age from 20 to 54 years. The etiologic factor was rheumatic fever in 8, syphilis in 2, and congenital malformation in one. All but one were in congestive failure at the time of operation Five had angina pectoris and 4 had calcification of the aortic valve. All had systolic as well as aortic diastolic murmurs. Further details are incorporated in Table I.

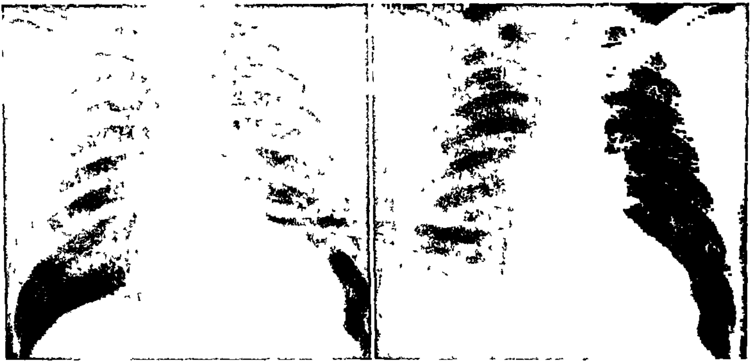

Fig 8.—Patient A P Cardiac silhouette before and one year after circumclusion for aortic insufficiency *Left,* Before operation, July 11, 1955 *Right,* After operation, July 9, 1956

DISCUSSION

Objective evaluation of this operation has been difficult. The technique must be modified according to the anatomic findings and cannot be rehearsed on animals. All but one patient operated upon were terminal. Among such a group of patients, a useful procedure can fail to prove itself. Fortunately, the first patient was conspicuously benefited An orthopneic, dying man in overt congestive failure, with angina and syncope in bed, was out of failure and free of pain and syncope when discharged on the tenth postoperative day. He returned to vigorous farming for one year. Reduction in cardiac size is illustrated (Fig. 8). Eventually, after 18 months, he succumbed to congestive heart failure secondary to a pulmonary infection. The first 12 postoperative months of rehabilitation were gratifying and prompted further trial of the operation.

The second patient had an aorta with a pyramidal base and the dissection was not carried deep enough to prevent upward migration of the constricting ligature. The right coronary artery was compromised and the patient died

TABLE I. CIRCUMCLUSION FOR AORTIC INSUFFICENCY—OPERATIVE EXPERIENCE

PATIENT	AGE	ETIOLOGY	LEFT VENTR. FAILURE	ANGINA	CALCIFICATION IN VALVE	DATE OF OPERATION	RESULT	REMARKS
1 A. P.	44	Syphilitic	+	+	+	7/27/55	Late death	Marked improvement for one year; transient failure at 12 months, responded to therapy; died 18 months later, congestive heart failure
2 L. R.	20	RHD	+	0	0	10/21/55	Operative death	Died 24 hours postoperatively; autopsy, suture compromised right coronary artery
3 R. G.	39	RHD	+	0	0	3/5/56	Operative death	Hufnagel prosthesis 3 years previously, followed by recurrent failure; died 15 days postoperatively, hepatic failure
4 P. S.	36	Congenital	+	0	0	3/26/56	Late death	Associated coarctation, aortotomy and digital control of circumclusion, wound dehiscence and aortic rupture; died 33 days postoperatively
5 W. P.	41	RHD	+	+	0	6/6/56	Operative death	Sudden death 20 hours postoperatively; suture well placed

PATIENT	AGE	ETIOLOGY	LEFT VENTR. FAILURE	ANGINA	CALCIFICATION IN VALVE	DATE OF OPERATION	RESULT	REMARKS
6 J. S.	44	RHD	+	0	+	8/14/56	Improved	Totally disabled by congestive heart failure preoperatively; postoperatively, working
7 A. K.	39	RHD	+	+	+	9/24/56	Improved	
8 H. P.	39	RHD	+	0	0	10/5/56	Improved	
9 W. G.	44	RHD	+	0	+	11/5/56	Improved	
10 J. G.	29	RHD	0	+	0	12/19/56	Improved	Only patient operated without failure, angina decubitus relieved
11 H. P.	54	Syphilitic	+	+	0	1/4/57	Operative death	Diffuse atelectasis; died 36 hours postoperatively

the day after surgery. This led to a more extensive dissection and the development of the double-tapered suture (Fig. 5), which has considerably improved the security of placement.

The third patient in this series was in cardiac failure and died 15 days after surgery in hepatic coma. He had had a Hufnagel valve placed 3 years previously.

The fourth death occurred 33 days after operation in a patient who had to be readmitted to the hospital because of dehiscence of his wound. The aortotomy also ruptured and the patient exsanguinated. Whether infection or the known liver disease was responsible for this total failure of healing cannot be established. Autopsy indicated that the ligature was well placed and did correct his aortic incompetence. An associated coarctation of the aorta may have been significant.

The fifth death occurred suddenly 20 hours after surgery. It was of the sudden unexpected type seen in patients with severe disease of the aortic valve and was presumed to be from ventricular fibrillation. At autopsy, the circumclusion was perhaps 3 mm. higher than the ideal position and had not been tightened sufficiently but it did not compromise the coronary arteries.

The sixth death was due to massive atelectasis and occurred on the second postoperative day. This patient had been hyperventilated with 100 per cent oxygen because of marked S-T segment depression during operation. Extubation and shift to nasal oxygen without nitrogen transition probably contributed to the atelectasis. The circumclusion appeared effective on direct inspection and when studied on the pulsator.

This high mortality rate seems less formidable when viewed as the result of salvage surgery in a developmental phase. Indeed the first death was 1½ years after surgery and the patient enjoyed total rehabilitation from a moribund preoperative state for a period of more than one year. The surviving patients have shown encouraging improvement.

Efforts are being made to find objective means of evaluating this operation. Cardiac output studies and dye dilution curves may provide objective data. No patient has been permanently relieved of his aortic diastolic murmur, but this is not a reliable criterion of the hemodynamic situation as discussed above. The clinical course and serial roentgenograms seem to constitute our best current means of assessment.

The fear that the circumcluding suture might erode through the base of the aorta seems unwarranted. This does not occur in dogs when the descending aorta is reduced to one half of its lumen with silk, nylon, or cotton material. It had not occurred in any of the patients presented here who died. This includes the patient who survived 18 months.

The dissection of the base of the aorta may at first seem a formidable procedure. A limited amount of practice, however, will convince one that this can be done if reasonable care is employed. *The division of the conus ligament* to expose the bases of pulmonary artery and aorta and the left coronary artery is a vital maneuver. Better suture material may be developed in the future but a very satisfactory one is available.* A knowledge of this dissection may be useful when valve replacement becomes practical.

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*Ethicon, Inc., Somerville, New Jersey.

This technique of circumclusion constitutes a reasonable corrective procedure which should carry an acceptable operative risk for patients not terminally ill with noncalcific or only slightly calcific incompetent aortic valves

SUMMARY

The clinical course, diagnosis, morphologic types, and hemodynamics of aortic insufficiency are discussed Principles involved in its surgical correction are reviewed Eleven patients with pure aortic insufficiency have been operated upon by the circumclusion technique Five are living and improved There were 4 operative deaths, and 2 late deaths All but one were in congestive failure at the time of operation The etiologic factor was rheumatic fever in 8, syphilis in 2, and congenital malformation in one

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(For Discussion, see page 230)

THE CLINICAL AND PHYSIOLOGIC CRITERIA FOR SURGICAL CORRECTION OF MITRAL INSUFFICIENCY

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A CONTINUING study to evolve methods for the proper surgical management of patients with mitral insufficiency has been in progress for 5 years.

Initial experimental work led to the successful clinical application of an effective operative technique for correction of mitral regurgitation 2½ years ago.* This was the first reported case in which correction of regurgitation was objectively documented.[1-5] From that date through March, 1957, 49 consecutive patients with severe degrees of mitral insufficiency have been examined.

In this presentation, preoperative, operative, and postoperative findings, as well as current results of management, are considered in an attempt to derive criteria for the selection of candidates for corrective surgery.

CLINICAL MATERIAL

Of the 49 cases included in this report, 7 were males and 42 were females. The ages were between 7 and 58 years. Forty gave a positive history of rheumatic fever or of definite related manifestations. The onset of clearly progressive symptoms varied from 3 months to 38 years prior to our investigation of the case. Progressive symptoms were present for an average of 7.72 years. Fifteen patients were totally incapacitated by their disease from 6 months to 15 years. All were on a medical program for control of congestive failure. This regime consisted of medication with digitalis preparations, mecurial and oral diuretics, as well as low sodium diets and marked restriction of activity for periods from 2 months to 29 years, averaging 7.8 years.

Twenty-six patients were in a state of decompensation at the time of admission to our hospital service. These proved to be cases of medically intractable congestive failure. Three patients had suffered peripheral embolic episodes.

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*Jan. 20, 1955.

Symptoms in order of severity and frequency were fatigue, dyspnea, palpitations, orthopnea, cough, edema, abdominal fullness or distention, and precordial pain. Two patients had been subjected to mitral commissurotomy, 1½ and 2 years before. Four had previous exploratory cardiotomy.

A systolic murmur at the apex, transmitted to the left sternal border and to the axilla, Grade 3 to 4 in intensity, was present in all cases. A muffled or absent mitral first sound was noted in 26 cases. The mitral first sound was intensified in 17 cases. A markedly accentuated second pulmonic sound was observed in 29 cases. A systolic murmur transmitted to the neck was detected in 5. One of these was proved to have a moderate degree of aortic stenosis. Another is suspected of having this lesion. An apical diastolic murmur was detected in 28 cases. Ascites and/or hepatomegaly were present in 29 cases. Dependent edema and pulmonary râles were evident in 9.

Routine laboratory work was not remarkable except for an occasional mild anemia. Eight patients may have been in a state of low grade rheumatic activity. However, C-reactive protein, antistreptolysin, and antihyaluronidase titers, as well as erythrocyte sedimentation rates, were not consistent.

Six patients were in normal sinus rhythm. Forty-three were in atrial fibrillation. Of the latter, 8 showed frequent runs of ventricular extrasystoles or bigeminal rhythm. Changes suggestive of digitalis effect, myocardial ischemia, or of pericardial activity were noted in 18. Ventricular rates varied between 50 and 96 beats per minute and averaged 76 beats per minute. Two patients showed evidence of atrioventricular dissociation.

Phonocardiograms routinely showed wide amplitude vibrations throughout systole on the apical tracings. Notable diastolic vibrations were seen in a few.

Radiologic studies included fluoroscopic examination and posteroanterior left lateral and both anterior oblique teleoroentgenograms. The findings can be condensed into three groups, arbitrarily divided according to heart size; there were 14 patients with massive cardiomegaly. The cardiothoracic ratio in this group ranged between 0.703 and 0.875, averaging 0.772. Fourteen patients had marked cardiomegaly with cardiothoracic ratios of 0.602 to 0.683, averaging 0.639. Sixteen patients had moderate cardiomegaly with cardiothoracic ratios of 0.464 to 0.595, averaging 0.518. Roentgenograms of 5 patients were not available for measurement. Four of these were operative deaths and one patient is living.

Right heart catheterization was performed in all but 3 patients and partial or complete data are available (Table I). Left atrial puncture was performed, transbronchially,[6] in 23 cases.

The most obvious clinical differentiation of cases in this series was based on the degrees of incapacitation and response to medical management. On this basis the series is separated into two categories. Twenty-six patients were in medically intractable congestive failure. Many were totally incapacitated. Twenty-three were in chronic congestive failure, but responded to medical management. A few were able to perform light tasks but showed fatigue on more than mild exertion.

TABLE I

CASE	AGE	RA	RV	PA	PVC	LA	LV	ARTERIAL	C.O.	PULMONARY ARTERIOLAR RESISTANCE	TOTAL PULMONARY VASCULAR RESISTANCE
1	28							125/75			
2	24		25/14	80/47	20/11						
3	27		30/ 6	35/ 5	14/11			125/65			
4	41	15/ 7		58/30							
5	19	9/ 2	75/–1	88/35							
6	28	2/–3	20/–5	28/10	16/ 5			125/62			
7	21	1/–3	31/–6	30/11	16/–1			98/57			
8	33	7/ 0		120/60*							
9	38		48/ 0	46/11	49/17						
10	33	10/ 6	80/ 2	76/36	29/15						
11	38										
12	38	3/–2	23/–4	20/ 4				130/60			
13	19	(9)	50/10	55/20	(16)			118/75			
14	29	5/–1	64/ 0								
15	7	3/–4	12/ 1	12/ 5	10/ 6						
16	33	4/–3	42/–3		48/15						
17	25	5/ 0	35/–5	30/ 5	12/ 0			140/80			
18	30	9/ 4	38/ 7	38/18		42/24	130/8	115/65	8.03?		196
19	31	9/ 2	34/ 5			58/25			3.75		348
20	52	10/ 6	35/ 5	35/17				140/48	4.8		384
21	31	14/10	60/ 0	60/20	30/18			120/64			
22	19	-3/–10	35/–7	40/ 6	15/ 6			124/76			
23	34		60/ 0	60/18	40/12	48/?					
24	22	13/ 5	92/ 5	92/44	52/22	60/15		140/84	2.3	975	1998
25	48	24/15	84/10	80/40	52/22	40/17		126/80	1.56	1120	1960
26	35	7/ 2	40/ 3	36/13	32/12	30/17		120/70	2.71	178	766
27	27	(7)	38/ 7	37/18	30/12	42/17		120/60	1.5	624	1338
28	35	5/ 0	45/ 2	45/22	12/ 5	10/ 0		136/68	3.24	160	800
29	18	10/ 6	48/ 8	48/30	35/15	50/10		100/60	2.25	400	1280
30	55	13/10	64/11	64/32	49/20	85/22		116/74	2.05	560	1760
31	17	10/ 3	70/ 6	70/35	22/15	40/24		120/52	3.30	880	1120
32	36	10/ 5	30/ 6	30/13	23/10	23/ 8		108/50	2.80	164	550
33	52	14/10	64/10	64/36	44/22	45/25		155/76	2.67	400	1200
34	47	catheterization contraindicated									
35	31		40/ 5	45/25	28/?	42/25		110/60			
36	29	10/ 5	28/ 7	28/10	23/18	24/12		128/64			
37	36	10/ 3	32/ 6	32/15	30/12			96/48	3.36	89?	500
38	47	8/ 5		30/12	23/9	26/12		120/68	4.44	110	419
39	58		24/ 7	20/10	16/10	35/12		160/88	2.25		490
40	26	15/ 6	45/ 7	45/25	35/16	40/17		100/64	2.6	320	990
41	25	10/ 5	40/ 7	40/22	38/12	44/15		120/70	3.8	212	522
42	32	15/10	42/10	42/20	35/16			118/64	4.86	100	468
43	33	18/ 4	43/12	43/25	42/26	50/22			2.85	351	420
Patients Who Did Not Have Corrective Surgery											
1	37	20/13	72/16					130/80	2.50		
2	29	3/–4	25/–6	34/–2	14/–2			105/48			
3	54	15/ 7	45/ 9	45/22	30/15	33/15		150/80	5.58	310	492
4	34	8/ 5	50/ 7	50/25	47/14			130/90	2.93	215	880
5	29			40/22	25/10	45/10		140/80	3.71	325	610
6	32	17/12	80/14	80/45	40/25			130/80	2.84	790	1624

RESULTS OF SURGERY

Twenty five medically intractable patients were subjected to surgery Definitive surgery could not be performed in 5 of these Two had such valvular damage that any known operative technique would have been futile In 3 other patients examination of the size of the cardiac chambers, the degrees of left ventricular dilatation, and their poor tolerance to thoracotomy indicated that they would not be improved by corrective surgery Three had previous vena cava ligation [8] In 2 this was done before exploration One patient was not subjected to further operations One patient of the intractable group had a vena cava ligation, followed 6 weeks later by corrective surgery and is included below

Twenty patients in intractable congestive failure were subjected to circumferential suture of the mitral annulus Three are living In one, regurgitation was due to rupture of chordae tendineae This case has been reported elsewhere [9] The patient is a 55 year old man who was totally incapacitated but who is essentially asymptomatic 11 months after surgery It was considered at operation that an excellent mechanical correction of the defect had been obtained One other patient is now 11 months postoperative Correction in this instance was less satisfactory Although he was not expected to survive, he did reasonably well during the first few months postoperatively No recent follow up is available The third living patient had vena cava ligation She is now 1 5 months postoperative and shows some improvement Correction of regurgitation in this case was satisfactory

Seventeen of the patients who manifested intractable failure are dead Mechanical correction of the regurgitant defect was excellent in 2, good in 5, fair in 2, and unsuccessful in 2 In 5 others, the degree of correction could not be ascertained because of general circulatory deterioration during the operative procedure Four of these were operative deaths (within one week after operation) One survived 8 months despite 65 minutes of ventricular fibrillation at surgery This patient is said to have improved slightly at first but subsequently deteriorated In 10 patients, satisfactory correction of the defect was known to have been obtained at surgery Three of these were operative deaths Seven survived one to 8 months postoperatively One of the survivors was improved physically but mental depression led her to commit suicide 6 months after operation

Autopsies were performed in 12 of the 17 patients in this group Eight of these hearts were studied on the pulse duplicator which demonstrated that 6 were satisfactorily corrected by the purse string operation The 2 others possessed the anatomically uncorrectable lesions just mentioned

To recapitulate then, of 20 intractable cases in which circumferential suture was performed, 3 patients are living Two of these are 11 months postoperative and one is 1 5 months Eight survived one to 8 months after surgery and 9 died within the first week In 16 patients who were operative or late deaths, substantial degrees of correction of the regurgitation were evident from operative observation, autopsy findings, and study on the pulse duplicator [10, 11] Indeed, it is possible that the regurgitation was totally eliminated in 3 of

This, however, cannot be proved. That correction was lasting was evident in all the late deaths which were autopsied. No corrective effect was observed in 2 and no information is available on one patient.

The causes of death were apparent in only 4 of the fatal cases in which satisfactory correction is known to have been obtained. One patient had ventricular fibrillation during preliminary exploration of the heart. Purse-string operation of the mitral valve was carried out in the presence of this arrhythmia in the hope of improving forward flow. However, all resuscitative measures were of no avail. Two patients had previous exploratory cardiotomy, and dissection through the dense adhesions in the transverse sinus led to uncontrollable hemorrhage when the suture was placed. One patient committed suicide. Three of the remaining 10 patients developed ventricular fibrillation at surgery. One could not be resuscitated, one survived 4 days after 45 minutes of ventricular fibrillation at surgery, one other lived 8 months. No definitive cause of death was noted in these 2 other than progressive circulatory failure. In the 7 other deaths no clear cause of death is known. All of these patients ran a course compatible with progressive heart failure. No significant arrhythmias were detected in this group although 2 patients died suddenly 4 and 5 months after surgery and either ventricular fibrillation or atrioventricular block was suspected. Coronary ischemia could be suspected but this is not clearly indicated by electrocardiograms which reveal mild changes compatible with digitalis effect, traumatic pericarditis, or mild degrees of ischemia. Serial tracings showed improvement with return to the preoperative state within one to 4 weeks postoperatively. Three patients developed a state of peripheral vascular collapse which was gradual in its evolution, intractable, and fatal in 4 to 6 days after surgery. Low grade rheumatic activity preoperatively and/or postoperatively was suspected in 4.

It is our impression that despite satisfactory correction of mitral regurgitation these patients died in a state of forward failure which was rapidly fatal in 3 and led to pulmonary congestion and combined left and right heart failure in 7.

Twenty-three patients were categorized preoperatively in the chronic congestive failure group on the basis of their condition and the response to medical management. Seven of this group are dead. Five were operative deaths, one from an incompatible blood transfusion. One died in ventricular fibrillation at surgery and 3 died 4 days postoperatively in a state of circulatory depression associated with terminal bradycardia in 2. An electrocardiogram was not obtained terminally and although available tracings do not show atrioventricular dissociation, it is strongly suspected that this derangement had occurred. In the third case, irregular ventricular tachycardia was present until death.

Two patients died in the late postoperative period. One died 3 months after surgery from an avoidable technical error which resulted in an infected thrombotic focus on a segment of the suture erroneously placed across the lumen of the left atrium at the atrioventricular ring. This case has been described previously[5, 13]. Another patient died 8 months postoperatively. Although the cause of death is not clear, the knot of the suture had eroded into the wall of the atrium and presented in the lumen. This may have been a source of emboli not detected at autopsy. This result is also avoidable. Autopsy was performed in all 7 of these patients and satisfactory correction of the regurgitant defect was substantiated.

Sixteen of this group of 23 patients in chronic congestive failure are living 3 months to 2½ years after operation. Mechanical correction of the lesion in these was interpreted as excellent in 4, good in 6, and fair in 6. In the first 4, no regurgitant jet was apparent upon completion of the operation.

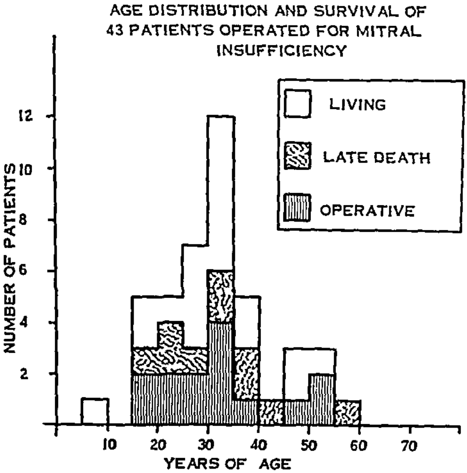
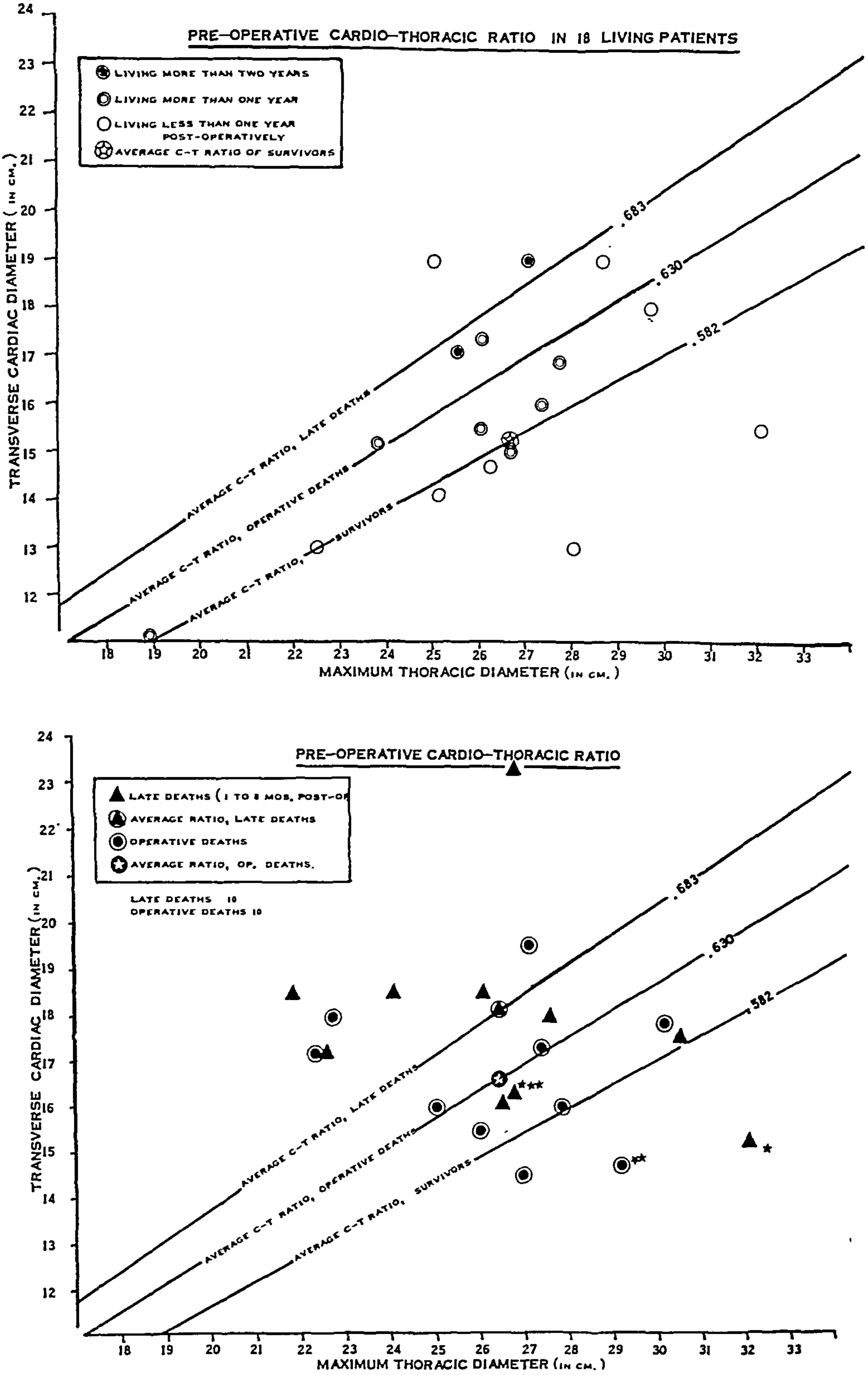


Fig 1

The murmur was totally abolished and remained undetectable for up to 2 weeks postoperatively. After this time, a systolic murmur of reduced intensity reappeared in all of these cases. All 16 patients have shown progressive and lasting improvement following surgery. Eleven of these individuals have returned to normal useful activity, either housework or gainful employment in their previous occupations. One of these patients had a preoperative embolic hemiplegia. Four are less than 7 months and one less than 10 months postoperative. This patient had suffered a preoperative peripheral embolus.

The postoperative course in this group has been extremely different from that of patients in the intractable failure group in spite of the fact that 4 of the survivors showed a picture compatible with low grade rheumatic activity pre or postoperatively.

Seven of the 16 had proved postoperative atrioventricular block within the first 2 weeks following surgery. In 2, digitalis intoxication could have been


PRE-OPERATIVE CARDIO-THORACIC RATIO IN 18 LIVING PATIENTS
LIVING MORE THAN TWO YEARS
LIVING MORE THAN ONE YEAR
LIVING LESS THAN ONE YEAR POST-OPERATIVELY
AVERAGE C-T RATIO OF SURVIVORS
AVERAGE C-T RATIO, LATE DEATHS
AVERAGE C-T RATIO, OPERATIVE DEATHS
AVERAGE C-T RATIO, SURVIVORS
.683
.630
.582
TRANSVERSE CARDIAC DIAMETER (IN CM.)
MAXIMUM THORACIC DIAMETER (IN CM.)
12 13 14 15 16 17 18 19 20 21 22 23 24
18 19 20 21 22 23 24 25 26 27 28 29 30 31 32 33
PRE-OPERATIVE CARDIO-THORACIC RATIO
LATE DEATHS (1 TO 8 MOS. POST-OP
AVERAGE RATIO, LATE DEATHS
OPERATIVE DEATHS
AVERAGE RATIO, OP. DEATHS.
LATE DEATHS 10
OPERATIVE DEATHS 10
AVERAGE C-T RATIO, LATE DEATHS
AVERAGE C-T RATIO, OPERATIVE DEATHS
AVERAGE C-T RATIO, SURVIVORS
.683
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.582
★★★
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TRANSVERSE CARDIAC DIAMETER (IN CM.)
MAXIMUM THORACIC DIAMETER (IN CM.)
12 13 14 15 16 17 18 19 20 21 22 23 24
18 19 20 21 22 23 24 25 26 27 28 29 30 31 32 33

Fig. 2.

responsible for this disturbance. In 5, the electrocardiogram had reverted to the preoperative rhythm by the time of discharge from the hospital (one month). In one case, that of the patient who died 3 months postoperatively with subacute bacterial endocarditis, the conduction disturbance persisted.

OBJECTIVE DATA

In the hope of formulating criteria for the application of corrective surgery for mitral insufficiency, the available data on the subjects of this study has been examined. The 43 patients operated upon have been grouped as follows: 19 patients who are living postoperatively, 14 patients who died one to 8 months after operation, and 10 patients who died within the first week of surgery. This eliminates the empirical preoperative evaluation made on clinical grounds. Unfortunately, due to the exigencies of clinical surgery, the information is not uniformly complete. A conscientious effort has been made, however, to gather all possible facts in every case and to subject these data to careful technical criticism.

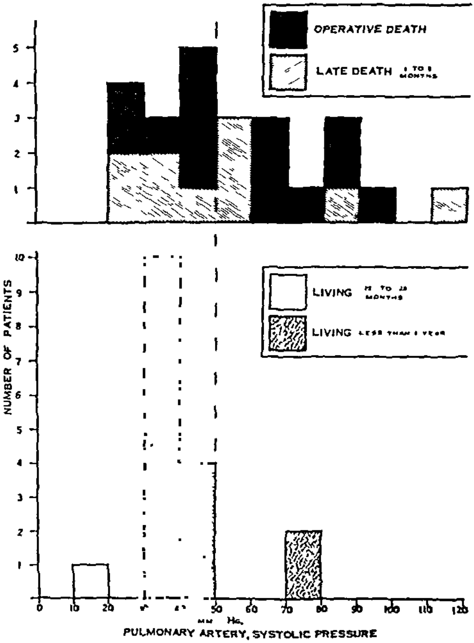
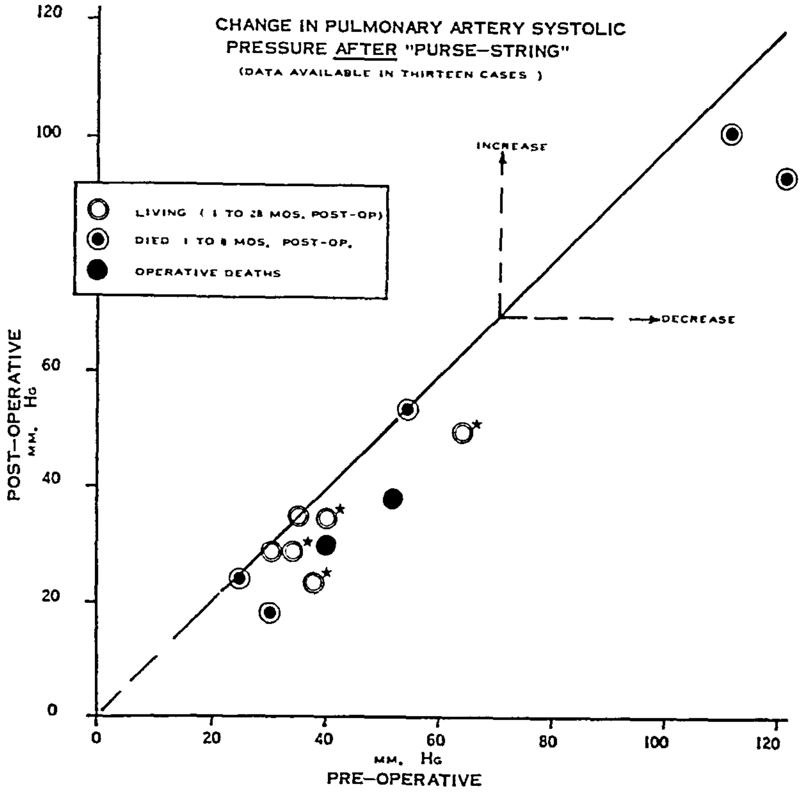


Fig. 3

The influence of age has been tabulated in Fig. 1. Insurance statistics compiled on a group of patients totaling more than 15,000 who had apical systolic murmurs interpreted as organic and who were followed for a period of



★ DATA FROM PRE- AND POST-OP. CARDIAC CATHETERIZATION AFTER 6, 8, 9 AND 12 MOS.

Fig. 4.

Fig. 5.—Extreme and average x-ray and hemodynamic findings in patients of the surviving group.* Left to right. *Top:* Cases 26 and 27. *Center:* Cases 28 and 40. *Bottom:* Cases 38 and 31.

		SURVIVAL†	PA	PVC	LA	LV	LV END DIAST.	ART.	CARDIAC OUTPUT (L./MIN.)	CARDIOTHORACIC RATIO
Average Heart and Average PA Systolic Pressure										
Case 26	Cath.	1 yr.	36/13	32/12	30/17			120/70	2.71	0.661
	Op.				30/12	104/3	8	112/68		
Cas 18	Cath.	1⅓ yr.	38/18	42/24	42/24	130/8		115/65	8.03?	0.669
	Op.				48/7	120/0	2	125/105		
Smallest and Largest Hearts										
Case 28	Cath.	11 mo.	45/22	12/15	10/0			136/68	3.24	0.464
	Op.				18/5	96/2	7	114/72		
Case 40	Cath.	3 mo.	45/25	35/16	40/17			100/64	2.6	0.663
	Op.				23/11	106/4	10	100/68		
Lowest and Highest PA Systolic Pressure										
Case 38	Cath.	5 mo.	30/12	23/9	26/12			120/68	4.4	0.561
	Op.				30/10	90/12	12			
Case 31	Cath.	10 mo.	70/35	22/15	40/24			120/52	3.3	0.683
	Op.				35/16	96/−4	0	104/60		

*Case 31 died at 1 year. All others are living and well at time of this publication.

†Survival time given at time of presentation (May, 1957).

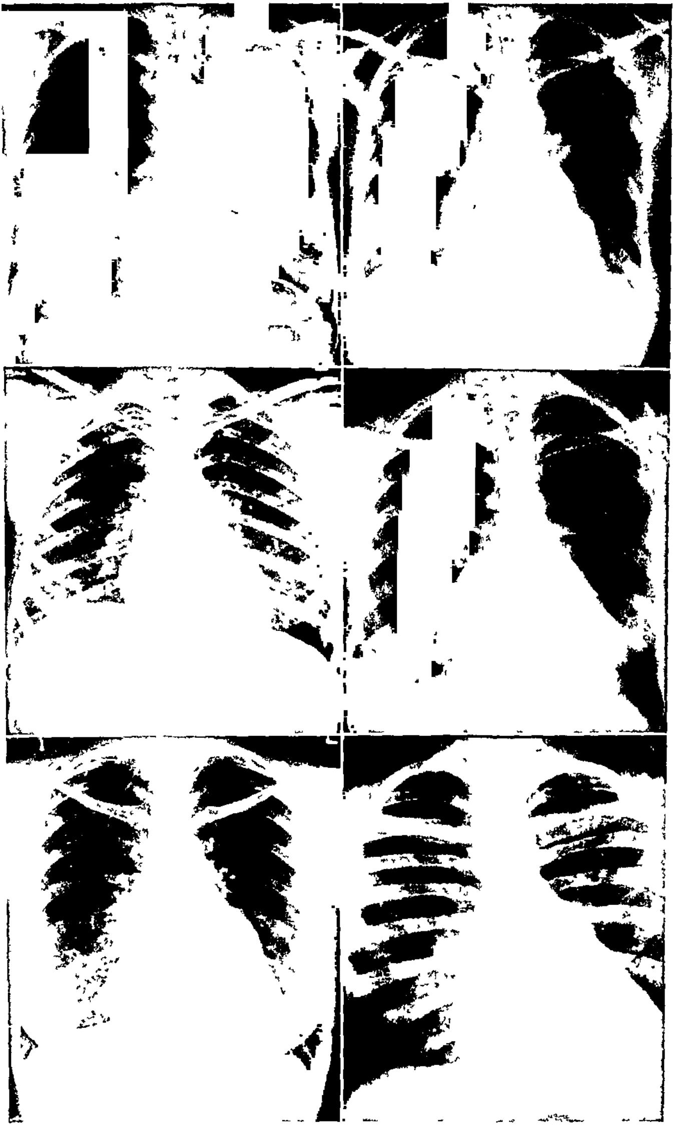


Fig 5.—*For legend see opposite page.*

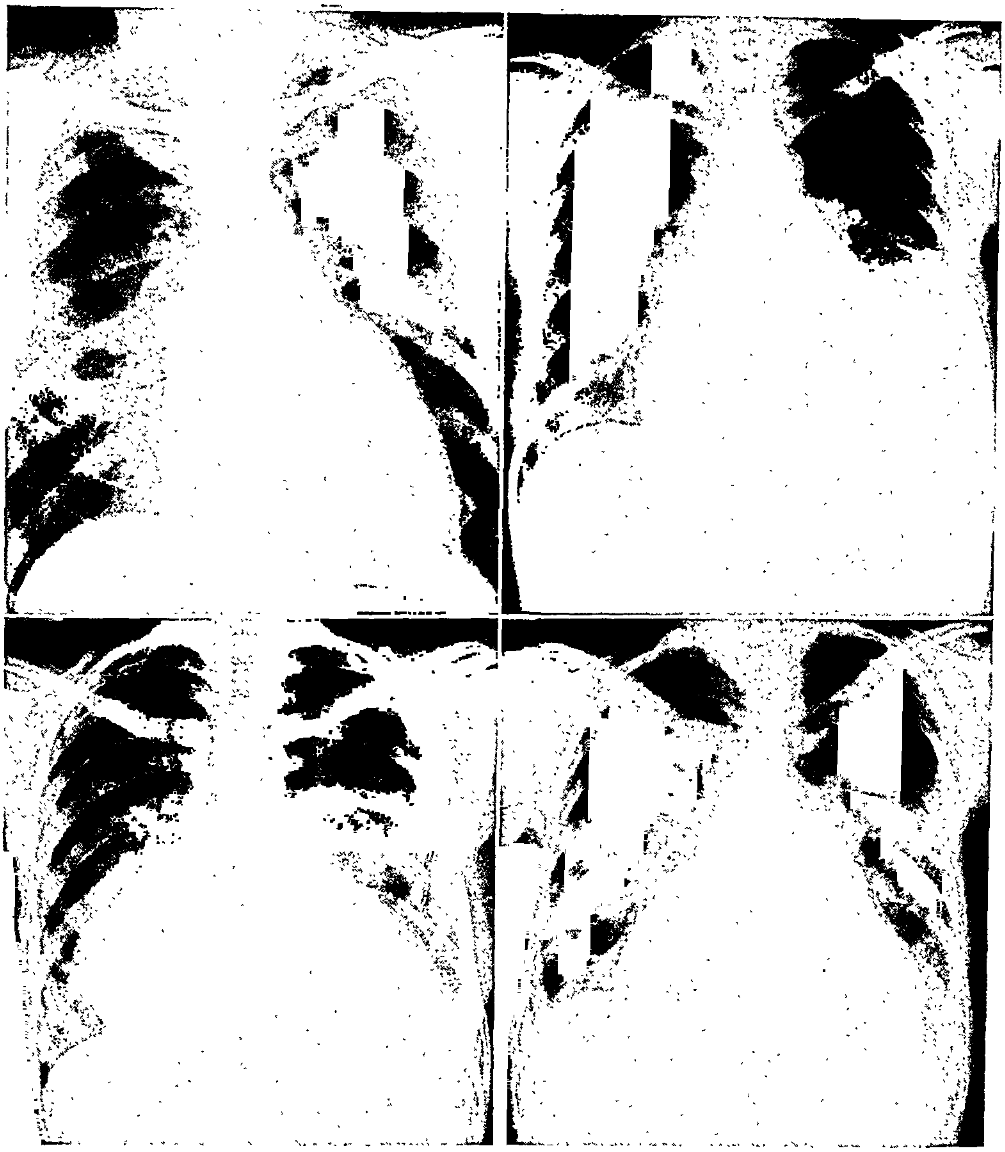


Fig. 6.—Extreme and average x-ray and hemodynamic findings in patients of the late death group. Left to right. *Top:* Cases 39 and 4. *Bottom:* Cases 6 and 8.

		SURVIVAL (MO.)	PA	PVC	LA	LV	LV END DIAST.	ART.	CARDIAC OUTPUT (L./MIN.)	CARDIOTHORACIC RATIO
Smallest Heart Lowest PA Pressure			20/10							
Case 39	Cath.	1½		16/10	35/12			160/68	2.25	0.578
	Op.				30/10	90/12	12			
Average Heart Average PA Pressure			58/30							
Case 4	Cath.	1	54/25							0.756
	Op.				28/11	155/−2	3	130/65		
Largest Heart			28/10							
Case 6	Cath.	2½	48/12	16/5				125/62		0.870
	Op.					80/−8	−4(?)	120/70		
Highest PA Pressure										
Case 8	Cath.	1½	120/60							0.712
	Op.					128/0	10	120/80		

15 years indicate the mortality risk of such individuals. Those who had, in addition to the murmur, moderate cardiac enlargement but gave no history of rheumatic fever had a mortality rate 7.6 times greater than the standard risk. Those who had a definite history of rheumatic fever in addition to the other findings were subject to a mortality risk 11.1 times greater than the standard. It it is assumed that this group largely represents cases of predominant mitral insufficiency, it is evident that we are dealing with a condition which is highly fatal in a relatively short time. The mortality percentage increases abruptly from less than 10 per cent at the 5 year follow up to 20 per cent at 10 years and nearly 35 per cent at 15 years.* The vast majority of our patients fall between the age limits of 15 to 40 years, the median being 30 to 35 years of age. This group of patients had definite progressive symptoms for an average of 7.7 years, indicating that their lesion perhaps including cardiomegaly was present for even longer periods of time. Their mortality risk then would fall within the limits of the insurance figures quoted above.

It was hoped that x-ray studies might provide a practical means to evaluate the stage of cardiac derangement. The average preoperative cardiothoracic ratio of the living patients is 0.582, that of the operative deaths is 0.630, and that of the late deaths is 0.683 (Fig. 2). Obviously the value of this information is of little significance by itself. It is of importance to note, however, that only 2 of the survivors had hearts larger than the average ratio of the late death group, while 5 of the operative deaths and 2 of the late deaths were above that figure. Also it is of interest that only 3 of the fatalities, one due to incompatible transfusion, and one a late death due to technical error, were below the average ratio of the survivor group.

A correlation of cardiovascular pressures and of heart size by x-ray examination has been attempted in these three groups. Fig. 3 shows the preoperative pulmonary artery systolic pressure in 17 of the survivors and in all of the fatalities. It is not surprising that only 2 patients with pulmonary artery pressures exceeding 50 mm. Hg systolic are living. Yet in 10 patients in whom the information is available the pulmonary artery pressure decreased immediately postoperatively. It remained unchanged in 3 (Fig. 4).

In Figs. 5, 6, and 7 are reproduced the preoperative posteroanterior roentgenograms of average and extreme cases of the living group, the operative deaths, and the late deaths. In the captions are listed the findings of cardiac catheterization, the operative pressures, the cardiothoracic ratio, and the length of survival. There are clear differences in the roentgenograms of the hearts in each group.

Although there is no true correlation between elevation of pulmonary artery pressure and heart size, it is apparent in Fig. 8 that the surviving patients were individuals in whom both cardiac size and pulmonary artery pressure show some relationship, whereas among the fatalities there was wide scatter.

Lung biopsy or autopsy in 22 of 40 cases showed definite pulmonary arteriolar damage in only 4. It is interesting to speculate that pulmonary hypertension in mitral regurgitation is perhaps of more acute onset than it is in mitral

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*From Bolt, W., and Lew, E. A.: Prognostic Value of Life Insurance Mortality Investigations, Exhibit at A.M.A. Convention, Atlantic City, 1955.

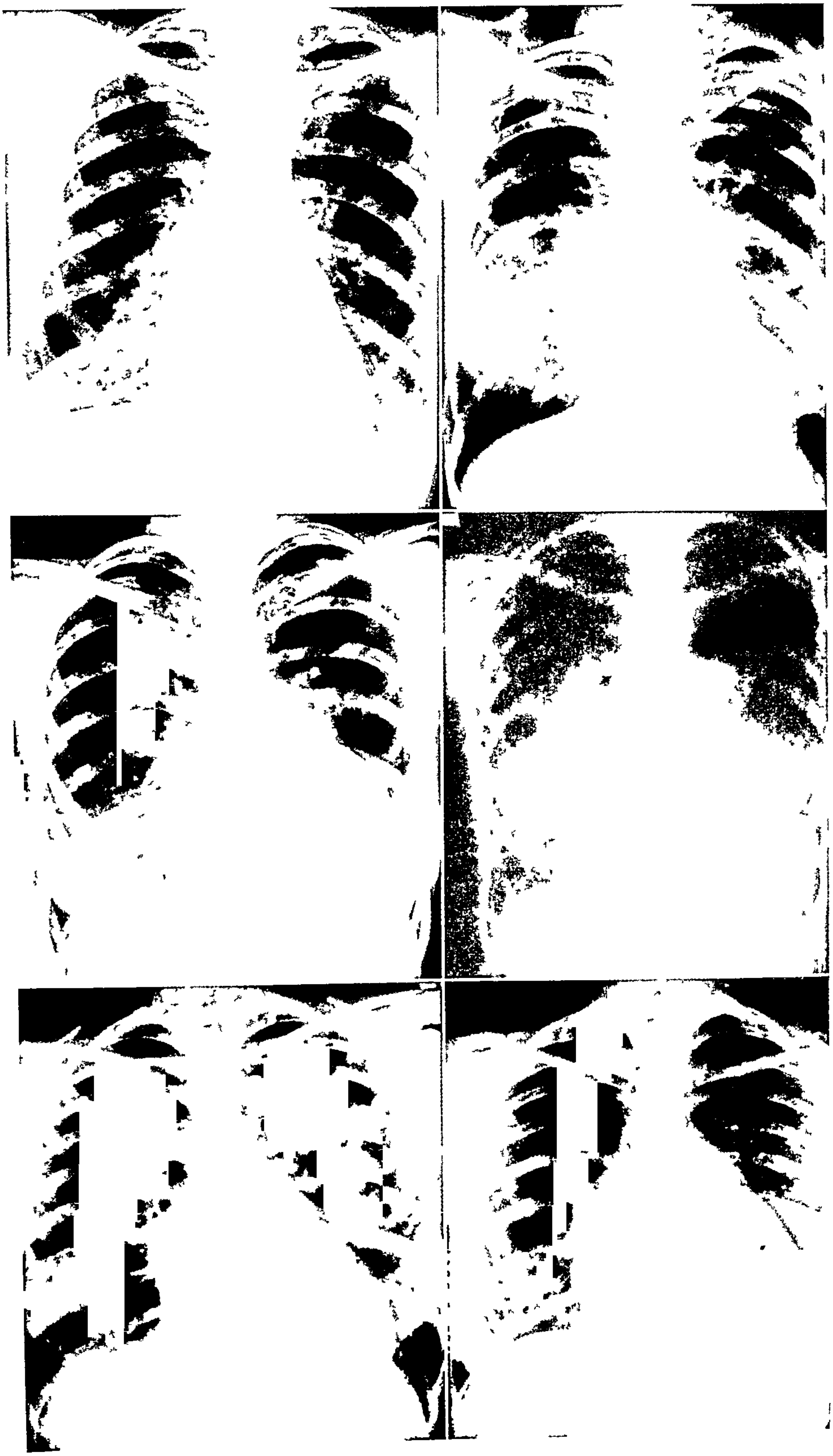

Fig. 7.—*For legend see opposite page.*

stenosis. Increased impediment to forward flow, concurrent with the inception of left ventricular failure appears to be more critical than the influence of the regurgitant defect per se in determining pulmonary hypertension. However, there does not seem to be positive correlation between left ventricular end diastolic and pulmonary artery systolic pressure.

The left ventricular end diastolic pressure is plotted in Fig. 9 to present the change in this parameter following correction of insufficiency. In 10 of the survivors it decreased significantly. In 4 it did not change (3 of these had essentially normal pressures initially). In 4 others, it increased. Among 14 cases of the fatal group, 7 show an increase. Five of these were operative deaths. Five showed a decrease, these were late deaths. One remained unchanged. None of the survivors had preoperative end diastolic pressures of more than 14 mm Hg. Two of the fatalities had higher pressures. The end diastolic pressure postoperatively in the survivors was above 10 in only 3 cases whereas, in the fatalities, 8 were between 10 and 20 mm Hg. Five of these were operative deaths. The end diastolic atrioventricular gradient in 40 of the 43 cases is plotted in Fig. 10. Postoperatively this information is available in only 31 cases, but it is apparent that there is little change in this measurement. The finding of numerous instances of higher left ventricular end diastolic pressure in the ventricle than in the atrium was unexpected but follows the pattern of grave left ventricular failure with marked increase in initial tension. Among the patients in whom there was a positive gradient, anatomic mitral stenosis was present in only 2. In several others there were varying degrees of commissural fusion of the leaflets but the effective orifice of the valve was ample (indeed, larger than normal in many). In 9 patients, commissurotomy was performed to improve valvular motion, in one to enlarge the effective orifice. The magnitude of regurgitant flow is a function of left ventriculo-atrial systolic

Fig. 7.—Extreme and average x-ray and hemodynamic findings in patients of the operative death group. Left to right: *Top*, Cases 20 and 14. *Center*, Cases 29 and 5. *Bottom*, Cases 36 and 24.

		PA	PVC	LA	LV	LV END DIAST	A T	CARDIAC OUTPUT (L./MIN.)	CARDIO-THORACIC RATIO
Smallest Hearts									
Case 20	Cath	35/17					140/18	4.8	0.536
	Op			35/10	110/-4	4			
Case 14	Cath	64/?							0.539
	Op	52/24		44/10	85/0	8	88/50		
Average Heart and Average P.A. Systolic Pressure									
Case 29	Cath	48/30	3 /1	?0/10			100/60	2.25	0.641
	Op			18/10	108/ 2	[illegible]	90/70		
Largest Heart									
Case 5	Cath	88/3							0.875
	Op	110/70		38/?	102/-5	18	110/80		
Lowest and Highest P.A. Systolic Pressure									
Case 36	Cath	28/10	23/18	24/12			128/64		0.576
	Op	40/20		35/16	110/4	10	112/74		
Case 24	Cath	92/44		60/15			140/84	2.3	0.659
	Op			60/18	120/7	8	124/84		

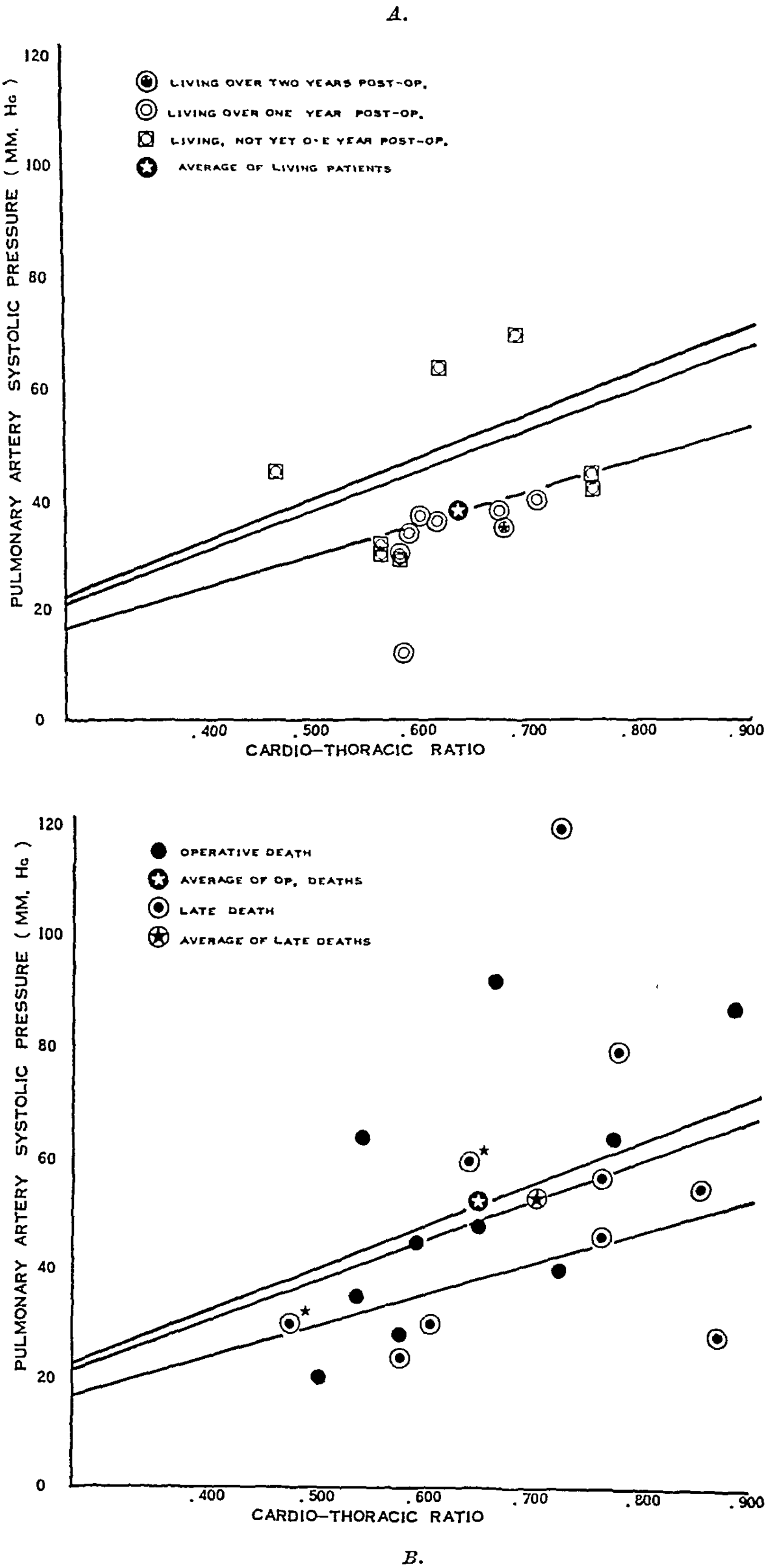

Fig. 8.—In the lower graph (*B*), the asterisks indicate one death by suicide (the uppermost symbol) and one due to technical error.

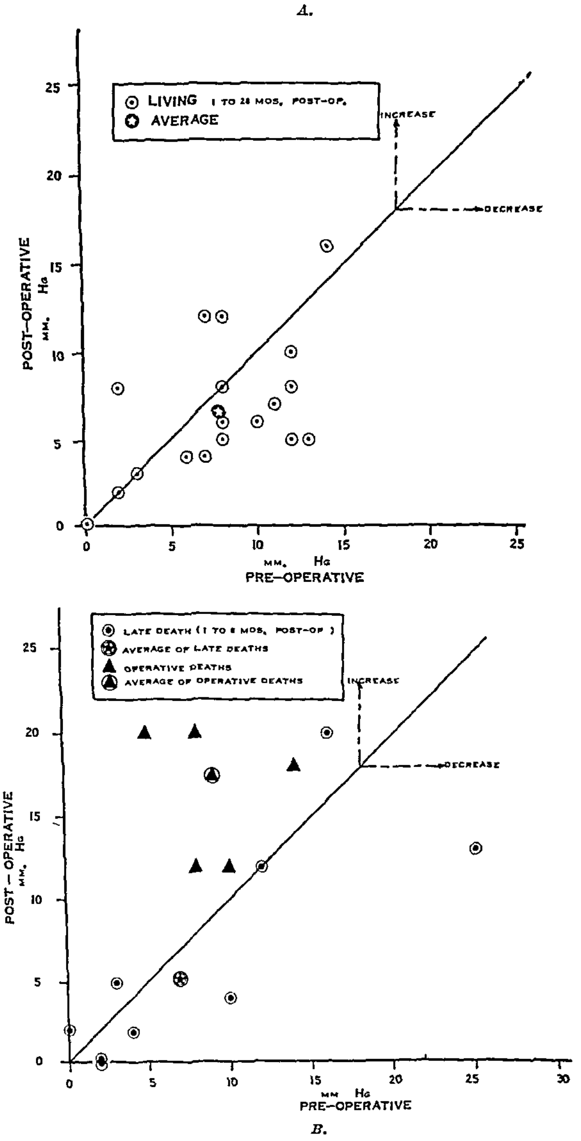

Fig 9 —*A*, Left ventricular end diastolic pressure in 18 living patients, before and after operation *B*, Left ventricular end-diastolic pressure in 14 patients (5 operative and 9 late deaths), before and after operation

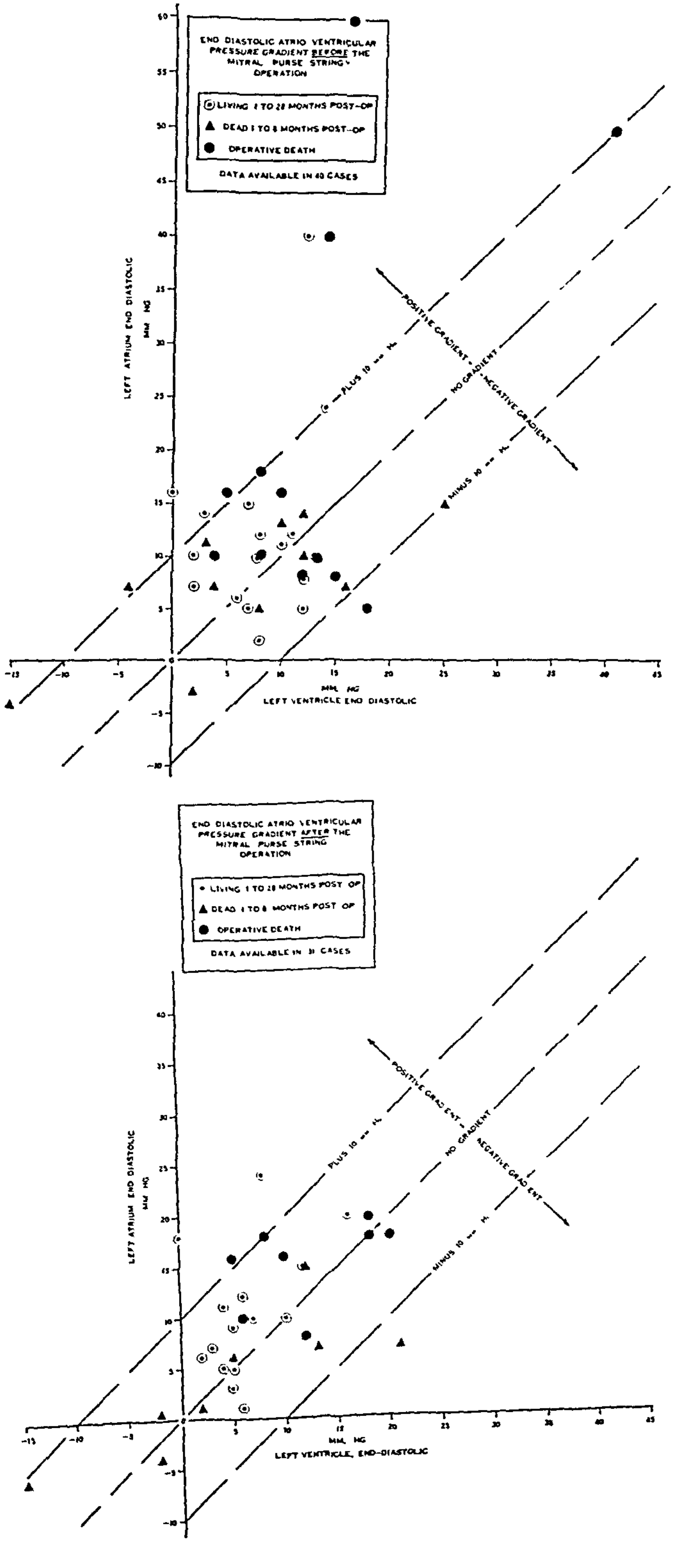
END DIASTOLIC ATRIO VENTRICULAR PRESSURE GRADIENT BEFORE THE MITRAL PURSE STRING OPERATION
⊙ LIVING 1 TO 28 MONTHS POST-OP
▲ DEAD 1 TO 8 MONTHS POST-OP
● OPERATIVE DEATH
DATA AVAILABLE IN 40 CASES
LEFT ATRIUM END DIASTOLIC MM HG
MM. HG LEFT VENTRICLE END DIASTOLIC
PLUS 10 MM. HG.
NO GRADIENT
MINUS 10 MM. HG.
POSITIVE GRADIENT
NEGATIVE GRADIENT
60
55
50
45
40
35
30
25
20
15
10
5
0
-5
-10
-15
45
END DIASTOLIC ATRIO VENTRICULAR PRESSURE GRADIENT AFTER THE MITRAL PURSE STRING OPERATION
• LIVING 1 TO 28 MONTHS POST OP
▲ DEAD 1 TO 8 MONTHS POST OP
● OPERATIVE DEATH
DATA AVAILABLE IN 31 CASES
LEFT ATRIUM END DIASTOLIC MM HG
MM. HG LEFT VENTRICLE, END-DIASTOLIC

Fig. 10.

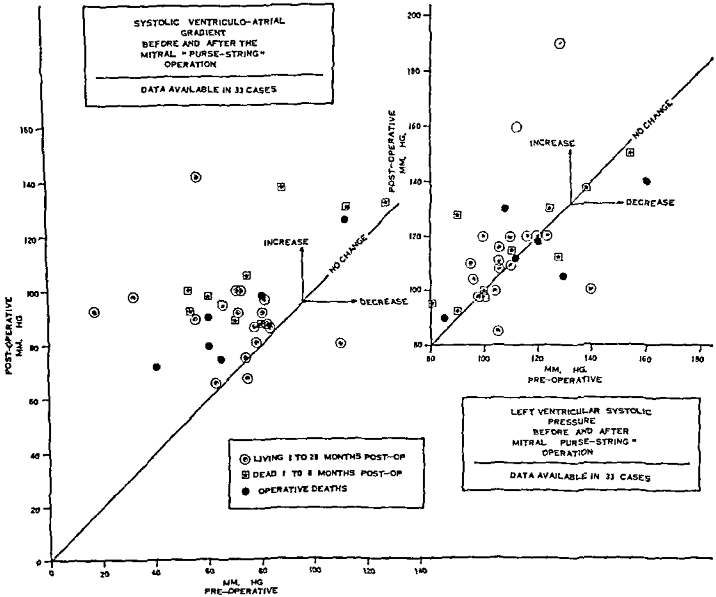
SYSTOLIC VENTRICULO-ATRIAL GRADIENT BEFORE AND AFTER THE MITRAL "PURSE-STRING" OPERATION
DATA AVAILABLE IN 33 CASES
POST-OPERATIVE MM. HG
MM. HG PRE-OPERATIVE
0
20
40
60
80
100
120
140
160
INCREASE
NO CHANGE
DECREASE
LIVING 1 TO 28 MONTHS POST-OP
DEAD 1 TO 8 MONTHS POST-OP
OPERATIVE DEATHS
LEFT VENTRICULAR SYSTOLIC PRESSURE BEFORE AND AFTER MITRAL "PURSE-STRING" OPERATION
DATA AVAILABLE IN 33 CASES
POST-OPERATIVE MM. HG.
MM. HG. PRE-OPERATIVE
80
100
120
140
160
180
200

Fig. 11.

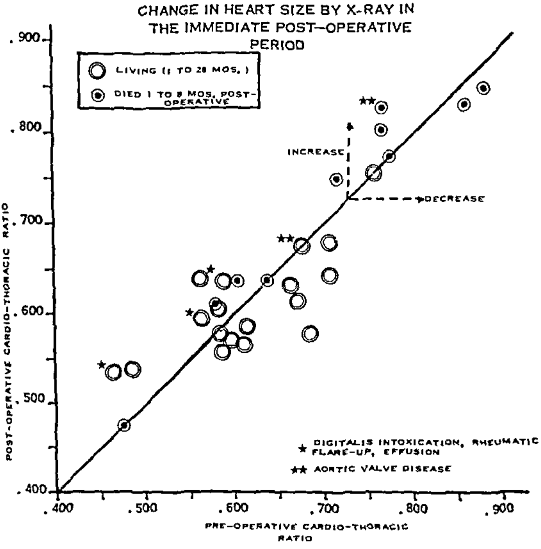
CHANGE IN HEART SIZE BY X-RAY IN THE IMMEDIATE POST-OPERATIVE PERIOD
LIVING (1 TO 28 MOS.)
DIED 1 TO 8 MOS. POST-OPERATIVE
INCREASE
DECREASE
.900
.800
.700
.600
.500
.400
POST-OPERATIVE CARDIO-THORACIC RATIO
PRE-OPERATIVE CARDIO-THORACIC RATIO
★ DIGITALIS INTOXICATION, RHEUMATIC FLARE-UP, EFFUSION
★★ AORTIC VALVE DISEASE

Fig. 12.

gradient and of the size of the regurgitant mitral orifice, as well as of duration of systole. In Fig. 11, this gradient is plotted before and after surgery showing a consistent rise indicating more efficient LV ejection forward against peripheral resistance by virtue of the fact that mitral valvular occlusive resistance has been increased. The gradient rise is partly due to left ventricular systolic pressure rise and partly due to left atrial pressure drop. No significant change in end-diastolic atrioventricular gradient was observed.

Postoperative change in heart size,* as evaluated radiographically, is summarized in Fig. 12.

The consistency with which radiologic reduction of the heart size is seen in the survivors having an uncomplicated postoperative course and the failure of the heart to decrease in size in the late deaths appears to be of some significance. Reduction of heart size in the former group is due obviously to the fact that regurgitation is reduced. In the others, cardiomegally remains unchanged despite reduction of regurgitation and can only be explained on the basis of loss of myocardial contractility.

DISCUSSION AND CONCLUSIONS

The preoperative state of these patients justifies considering at least half of them grave operative risks. On this basis a high operative mortality is to be expected. However, if the "purse-string" operation corrects mitral regurgitation then one would expect good results in the survivors. There is ample evidence that the mechanical defect of mitral insufficiency is corrected by this procedure in the vast majority of the cases. Indeed, patients in less advanced stages of the disease obtain highly satisfactory results. It is, however, the fate of the severely ill patients in whom correction is attained and who survive surgery that is most disconcerting.

It appears that the outcome, in cases like these, may be determined by one of two circumstances: either surgical trauma to the heart cannot be tolerated, or the dynamic changes due to the correction of the regurgitant lesion are such that the gravely damaged heart cannot adjust to them.

In a small number of patients, operative damage to cardiac structures has become evident. Instances of this have been mentioned in which conduction disturbances are demonstrable. The changes are transient, disappearing in a short period of time. Frequently, as in other types of cardiac surgery, electrocardiographic changes compatible with digitalis intoxication, traumatic or rheumatic pericarditis, or myocardial ischemia of minor degrees have been observed. These also are transient changes and serial tracings consistently show reversion to the preoperative state. The possibility of even slight degrees of myocardial ischemia cannot be disregarded, however, and efforts are made to avoid this if such is due to surgical injury. Be that as it may, the operative survival of 29 of 43 patients representing grave surgical risks at the outset would suggest that surgical trauma, well tolerated by the long-term survivors, is of less importance than the physiologic derangement which leads to late death in the most advanced cases.

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*Although such a comparison based on the cardiothoracic measurement is subject to the fallacies of this ratio when it involves different cases, it seems less objectionable when it is used to compare individual changes, particularly when the films are made by the same technique in the same institution.

The dynamics of mitral insufficiency are such that the major cardiac compensatory mechanism must be ventricular dilatation. Increased ventricular volume to accommodate forward stroke volume, regurgitant stroke volume, and residual volume implies extreme degrees of myocardial fiber lengthening. Evidence from animal and in vitro experiments and from clinical measurements indicates that these volumes are considerably larger than has heretofore been thought. It seems likely that in cases, such as some of those here presented, myocardial fiber length has exceeded elastic tolerance and recovery of contractility is not possible. This phenomenon might be compared to the fatigue sustained by inert materials when their tensile or elastic limits are overreached.

In the heart with mitral insufficiency in which the limit of myocardial tension or stretch has not yet been reached, correction of the regurgitant defect results in the following changes: immediate increase in cardiac output, rise in ventricular and aortic mean and systolic pressures, reduction in left ventricular and atrial size (thus, decrease in residual volume), decreased left atrial pressure, and decreased left ventricular initial tension (end-diastolic pressure). The duration of "isometric" or "pre-ejection" phase as well as in that of maximal ejection is observed to increase both in experimental animals and in patients. Heart rate is reduced. All of these changes indicate improved myocardial function. It appears likely that correction of regurgitation results in an increase of effective cardiac work. If this is true, there is also an increase in efficiency of muscular contraction because of shortening of fiber length.

In the far-advanced stages, however, loss of myocardial contractility by, literally, "overstretching" makes impossible reduction in fiber length. Correction of the regurgitant defect increases forward flow and decreases regurgitant flow but may leave residual volume unchanged or even increased. All changes indicative of increased forward flow and reduced regurgitation occur as in the previous group. However, in many of these cases, initial ventricular tension is increased and maximum ventricular systolic pressure may be decreased, even though there is demonstrated a rise in ventriculo-atrial systolic gradient. These changes again imply increased work of the heart, however, contrary to the favorable reduction in muscle fiber length which occurs in the previous group, in these, reduction of ventricular volume (shortening of fiber) fails to occur. Myocardial efficiency then must not improve and, indeed, may deteriorate.

If this hypothesis proves to be correct it poses the problem of either finding ways by which myocardial contractility may be enhanced or of perfecting our ability to select surgical candidates, eliminating those in whom the elastic reserve of the myocardium is exhausted.

Observations that ligation of the vena cava below the renal veins in dogs[7, 8] protects them in the acute phase of experimentally produced mitral regurgitation have led to application of this procedure in 5 severely ill patients. Four of these were cases of intractable mitral insufficiency. The other was a case of interatrial septal defect with right-to-left shunt. All have shown some degree of early improvement. One of the patients with insufficiency had corrective surgery 6 weeks after vein ligation and has progressed satisfactorily. Fig. 13 illustrates the change in heart size seen in one such patient. The rapid re-establishment of venous collateral circulation appears at this preliminary stage to render this procedure of little palliative value. However, if reduction in heart size is attained in such advanced cases, this might indicate a residual capacity of the myocardium to recover contractility. Inferior cava ligation, then, may be one means of preparing the "intractable" patient for corrective surgery, or it may aid in selecting those patients who may benefit from corrective surgery. It is too early to make any definitive conclusions in this regard.

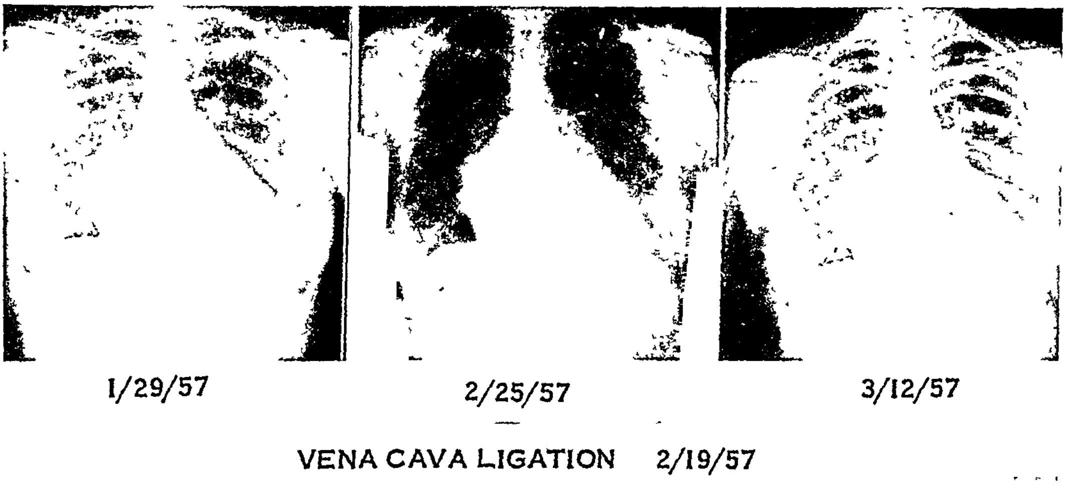

Fig. 13.—Change in heart size following vena cava ligation in a patient of the medically intractable group.

It is apparent that none of the methods used suffice to evaluate the anatomic state of the valve or the functional reserve of the myocardium in mitral insufficiency. Knowledge of both is mandatory if we are to avoid subjecting many patients to fruitless surgery. Fortunately, in pure or predominating mitral insufficiency, the valve is infrequently beyond restoration to adequate function. In this form of regurgitation, however, the muscle is very often irreparably damaged. This represents the greatest problem in the selection of the surgical candidate. Expert interpretation of the ballistocardiogram might offer some useful information. Further experience with the electrocardiographic interpretation of ventricular overloading might similarly be of value. These two techniques have not been adequately investigated with this purpose.

Many workers, ourselves among them, have been engaged in efforts to find a reliable means of quantitating regurgitant volume in the hope that this will prove a useful "yardstick." Our experience with various methods has been disappointing. Direct measurement of regurgitant flow in animals is impractical and unreliable. Direct measurement in vitro, using autopsy hearts on the pulse-duplicator, has yielded figures for regurgitant flow of some two to four times forward flow.[12] This, however, has only indirect application to clinical practice. Since, in patients, direct measurement is highly impractical and dangerous, indicator dilution studies are currently the most promising means of obtaining such data. Preliminary work in this regard has produced some interesting but inconclusive results.[12] An example of such a study is illustrated in Fig. 14. However, the problem of fractionating the measurement of central dilution to separate quantitatively not only forward flow from central volume but also left ventricular and atrial residual volumes from regurgitant flow remains to be solved. This is of particular importance when cardiac size influences dilution to the degrees that it does in serious mitral insufficiency. For this reason, at the present time, serial injections of Evans blue dye into the ascending aorta, the left ventricle, and the left atrium afford only partial information.[12]

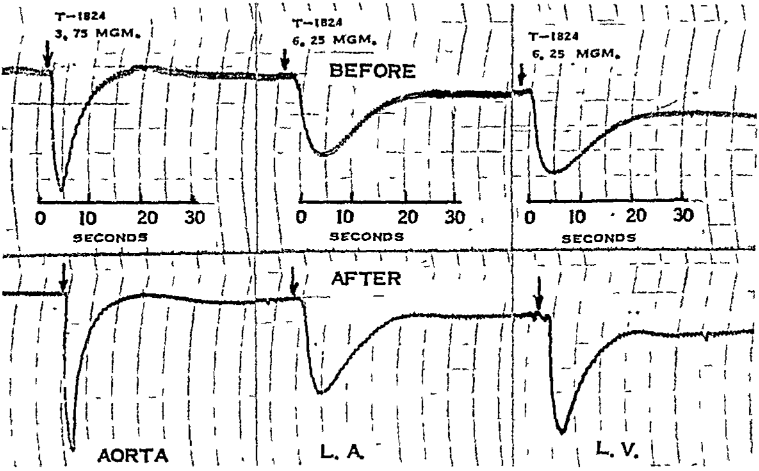

Fig. 14.—Indicator dilution curves of serial injections before and after correction of regurgitation. Although after correction of the lesion, cardiac output, calculated from curves of the aortic injection, is more than tripled, the change in the curves of ventricular and atrial injection can only be evaluated in terms of total central volume without specific fractionation into residual and regurgitant volumes. In this instance, regurgitation was markedly diminished but not abolished, change in ventricular size was also clearly seen.

The inconsistency of the usual objective data has been such that selection of patients for surgery remains difficult and uncertain. It is now known that in certain patients with extremely high pulmonary artery pressure, enormous hearts, and markedly elevated left ventricular end-diastolic pressure, corrective surgery alone, no matter how effective mechanically, is contraindicated. There is, however, much overlap in these findings among patients who have obtained the greatest or the least benefit. Other data such as ventricular strain patterns on electrocardiography, pulmonary wedge or left atrial and left ventricular pressure pulses is useful only in making the qualitative diagnosis of mitral insufficiency which, in almost every instance in this series, was clinically obvious initially.

The clinical history is of little, if some, help in arriving at a surgical decision. Patients with the longest history of progressive incapacity generally represent the greatest risks. However, a few individuals with cardiomegaly of neither extreme but with histories of more than 10 or 15 years of symptoms do well. Others with enormous hearts but only recent onset of severe incapacity also do well. The response to medical management exhibited by most of these patients has been uniformly unsatisfactory in the intractable group and devoid of significant features in the other group.

The lessons learned at operation have been of the greatest value. It is now quite apparent upon examining some hearts that tolerance to surgical manipulation is out of the question. Others impress one with almost complete certainty of success. Recent experiences have borne out these impressions. The over-all size of the heart is of relatively little importance except when there is obvious left ventricular dilatation. This is frequently accentuated on opening the pericardium. The appearance of such hearts suggests the shape of a Rugby ball. In these, the muscle is thin and tense, pale or mottled. It is apparent to the eye and hand that contraction is weakened and of short stroke. Rhythm is unstable. Slight superficial stimuli and manipulation induce runs of bigeminal pulse, idioventricular beats, or even ventricular flutter or fibrillation. The patients' circulatory balance is precarious under anesthesia and supportive measures are almost routinely necessary.

In patients that do well, the heart has the contour of an acorn. The left atrium may be enormous but the ventricle is relatively small, pink, contracts strongly, and can be seen to "shrink" in systole.

It is unfortunate that, up to the present time, the final decision of whether or not corrective surgery for mitral insufficiency is compatible with recovery and benefit has necessitated thoracotomy in patients who fall in the mid-zones of objective preoperative evaluation. It is obvious that regardless of other circumstances and of the surgical methods used, if valvular tissues have undergone severe destruction, restoration of existing valve elments to function is impossible. On the other hand, with a myocardium which has not been damaged beyond its tolerance, if leaflet structure is available, retains adequate area and flexibility, and can be mobilized, there is no doubt that reduction of the size of the annulus restores, according to the individual circumstances, the maximum possible degree of valvular competence and reverses the insidious dynamic process of mitral regurgitation.

SUMMARY

Circumferential suture of the mitral valve has been used to correct mitral insufficiency in 43 of 49 patients in far-advanced stages of the disease.

A summary of objective and clinical findings preoperatively, at surgery, postoperatively, and of the results obtained is presented.

An attempt is made to correlate objective data available with the results of surgical management

It is concluded that

Pure or predominant mitral insufficiency can be corrected mechanically provided valvular area, mobility, and flexibility are retained

Despite satisfactory correction of the regurgitant lesion, lasting benefit cannot be expected in cases where myocardial contractility is destroyed by compensatory overdilatation

When valvular damage is excessive and myocardial exhaustion has supervened, surgical intervention, regardless of the method used, can be of no value

If surgical treatment is to be successful in the advanced stages of the disease, means must be sought to improve myocardial function Vena cava ligation may be of some value in this regard

Proper selection of cases should make possible the reduction of operative mortality, the elimination of late deaths, and should lead to the attainment of consistent and highly satisfactory results

Current methods of preoperative study, both clinical and objective, are inadequate to evaluate the state of the myocardium

The size of the heart by x ray examination, the degree of pulmonary hypertension, and the left ventricular end diastolic pressure are the only parameters which correlate in any manner with the results of surgery

The clinical and operative findings, particularly the latter, are currently our most useful guide to prognosis

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DISCUSSION

(PAPERS BY TAYLOR [PAGE 192], DAVILA [PAGE 206] AND THEIR ASSOCIATES)

DR. BENSON B. ROE, San Francisco, Calif.—Dr. Taylor and the group at the Peter Bent Brigham Hospital, as well as Dr. Lillehei, Dr. Swan, and many others, are certainly to be congratulated on the excellent results in their carefully selected cases with these ingenious methods and on taking advantage of open heart techniques for the approach to the aortic valve in correction of regurgitation. I believe, however, that everyone will agree that the extensive disease that takes place in many of these valves destroys the function of the valve which, in many instances, will never respond to reconstructive techniques and can be corrected only by the insertion of a suitable prosthesis. This challenging problem has been of interest to me for several years and, after many changes of design and many details of construction which time does not permit discussing, I would like to present to you our presently used valve.

This is a prosthetic valve made of a silicone pure material which is flexible. It is made cylindrical in shape, is introduced through a distal aortotomy, and its long axial length maintains its position as it is pushed over the aortic arch to be sutured in place in the ascending aorta. The ring which you see is folded in place and springs back into shape as it reaches the part of the aorta in which it will fit. This is the cut-off valve to show how it can be used in the various direct techniques.

Another design of the valve can be used for subcoronary placement. This merely shows that the valve is competent with only a small amount of leakage, and in the next strip of film you will see that it does leak a small amount of water at high pressure, but what you see at the top is mercury, not water; 300 cm. of water does not blow it out.

This is a film showing the valve with an artificial pumping mechanism to demonstrate that the cusps work. You notice that the upper commissure, for which I must apologize, was not properly prepared and therefore the two upper cusps do not retract properly. This valve was originally reported to be used in polyvinyl material which eventually lost its elasticity, which this material apparently will not do.

DR. F. HENRY ELLIS, JR., Rochester, Minn.—The concept of circumferential suture of the mitral valve ring is an inviting one. Several years ago, when Dr. Davila and Dr. Glover first presented this method before this Association, it appealed to me as being as logical and as promising a method for correction of mitral insufficiency as had yet been suggested. Because their experimental work was done primarily on dogs with acute mitral insufficiency, it seemed wise to us to try to test the method in dogs with chronic mitral insufficiency, a condition more comparable to that seen in man.

Dr. Keykendall, working with dogs in our laboratories, has been able to produce mitral insufficiency which closely resembles that seen in the human being; in other words, dilatation of the mitral ring developed in these dogs, and an enlarged left atrium, and exercise tolerance and so on, were noted. A group of these dogs underwent correction of the mitral insufficiency, and one of the methods used was circumferential suture of the mitral valve ring. It was possible, in 6 of these animals, to abolish completely the mitral insufficiency as determined by disappearance of the regurgitant jet at the time of operation. However, in 5 of 6 animals so treated, within 2 to 6 months after operation, there was a recurrence of mitral insufficiency as evidenced by return of the murmur and the presence of a regurgitant jet at the time of re-exploration.

In no case had the suture cut through into the atrium. However, it had loosened, as it migrated centrally in the tissues of the heart, to lie subendocardially. This experience has discouraged us from applying this method in human beings.

I should like to ask Dr. Davila two questions: Has he used this method in dogs with chronic mitral insufficiency? How often has he seen evidence of the recurrence of mitral insufficiency in patients surviving operation for any length of time?

DR CHARLES P BAILEY, Philadelphia, Pa —[The corrected discussion by Dr Bailey has not been returned by the discusser —EDITOR]

DR WARREN J TAYLOR, Boston, Mass (Closing) —I wish to thank the discussers for their comments

We too have been concerned, as have Dr Roe and his associates, with the limitations imposed by the nature of the pathologic valves themselves The ideal treatment is replacement of the diseased aortic valve by a prosthesis, positioned proximal to the coronary arteries

Dr Bailey's comments are pertinent At one time he made the observation that the base of the aorta spreads and does not taper Hence, a suture placed about such a cone might tend to "ride up" and threaten the coronary vessels We have tried to stress the importance of carrying the dissection proximal to the coronary ostia and *below the widest girth of the sinuses of Valsalva* In our experience, the base of the aorta has always tapered below this point A suture placed at this level will "ride down" away from the coronary arteries

We hope that a second point of difference is appreciated The circumcluding suture is placed *only about the base of the aorta* Widespread tissue compression, as occurs with the 'Sash Operation," is therefore avoided

DR JULIO C DAVILA, Philadelphia, Pa (Closing) —With regard to Dr Ellis' question, I would like to say that we do have evidence that patients who have had marked correction of their mechanical valvular defect, even to the abolition of the thrill and murmur, within a matter of a few weeks do show recurrences of the murmur This is, however, of a mild degree as compared to the preoperative state We have one patient in whom left atrial pressure is higher several months following correction We do not know whether this means there is more regurgitation She is doing very well

It is extremely important to understand that when the suture is placed it must be adjacent to the annulus (slide) This is a section of the mitral ring showing the lateral myocardium, the atrial wall, the base of the valve, the periannular fat pad, the circumflex coronary artery and the cardiac veins This is the fat pad of the atrioventricular group The suture is not placed in the myocardium It is placed beneath the epicardium in the fat pad When tying the suture it slides over the hump of ventricular myocardium and finds its place in close relationship to the ring If on tying the suture, it does not come to this position, it will gradually find its way there and therefore will result in some degree of loosening Evidence collected from a few other patients who have been catheterized, some after long periods of time following the procedure, show that despite the fact that they may have a persisting murmur, their increased cardiac output and their improvement in hemodynamic measurement indicate significant improvement in the regurgitation

With regard to the statement about whether or not the principle of annular reduction is going to be an adequate solution to this problem, this remains to be seen Open heart surgery may give us, perhaps, a better way to operate on these cases It seems, as I said before, that on the basis, not of theory but of repeated demonstration anatomically and physiologically, experimentally and clinically, that this principle is applicable until the time when we can replace the valve, because it does afford the maximum use of what natural valvular elements are available and it does not injure essential valvular structure It also causes a relaxation of leaflets, allowing better motion

Any technique which reduces the annulus, whether partially or completely, will restore valvular competence to a degree commensurate with the amount of valvular occluding elements available and with the extent to which these elements are mobilized and spared surgical injury Any injury to these valvular structures, on the other hand, will result in unpredictable degrees of distortion and mechanical dysfunction

I would like to thank the discussers for their comments

CORONARY HEART DISEASE

I. Hemodynamic Principles and Their Therapeutic Applications

B. L. Brofman, M.D. (by invitation), and Claude S. Beck, M.D.
Cleveland, Ohio

DESPITE the most optimistic wishful thinking to the contrary, coronary heart disease continues to be responsible for at least one third of all deaths in the United States,* and may actually be showing a real (rather than merely apparent) increase. The enormous destruction of economic productivity defies statistical analysis. Consequently, with the development of improved methods, there has been a justifiable renewal of interest in the surgical treatment of coronary heart disease. However, lest such efforts be ill advised, particularly in view of the indiscriminate application of such terms as "direct" and "indirect" surgical operation, it would now appear appropriate to attempt to establish a critical definition of the problem and to present a reappraisal of certain fundamental (and often disregarded) principles which determine coronary blood flow to the myocardium.

Although many of the observations and concepts herein presented may appear de novo and perhaps controversial (and are indeed so presented as to stimulate further critical reappraisal), it is pointed out that this presentation is based upon a remarkable experience which encompasses many thousands of laboratory experiments on the coronary circulation, hydraulic model experiments, operations on the heart in more than 300 patients with coronary heart disease, and at least 1,000 other heart operations for various noncoronary conditions. Of profound importance is a unique experience not heretofore available, in patients with coronary heart disease, for direct observations on such hearts. These observations included direct electrocardiographic recording from various epicardial, intramyocardial, and endocardial areas, and extensive hemodynamic studies, including measurements of coronary blood flow, and myocardial utilization of oxygen and various substrates. Thus, we have availed ourselves of a remarkable opportunity for a direct correlation of clinical, experimental, surgical, and pathologic observations.

Certain clinical phenomena associated with a priori reasoning were subjected to laboratory experimentation and, when not substantiated, were then rejected. Of greater importance, clinical observations confirmed by laboratory studies have resulted in the establishment of new concepts which provide a more

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*Latest available statistics from the National Office of Vital Statistics.

logical basis for the therapeutic approach to coronary heart disease. In the constant soul-searching attendant upon such experience, we frequently rejected certain clinical observations which seemed inconsistent with established concepts; however, the constant reproducibility of such observations eventually forced re-examination and ultimate modification of such concepts. Indeed, as recently stated in Lancet, it is hoped that this work “has thrown fresh light on old well-established theoretical concepts.”[1]

In the interests of a comprehensive presentation, and yet respecting the limitations of space, much of the material herein presented is necessarily brief, with no attempt at complete elaboration, which we will hope to achieve in subsequent publications.

Its very nature is such that clinical observation is the sine qua non evaluating the treatment of coronary heart disease. But this disease continue to defy “objective” evaluation. Consequently its treatment has suffered from a great reluctance to make the necessary correlations between clinical and experimental observations. The historical development of present-day comprehension of coronary heart disease is characterized by a disposition to marvel at, and then reject, reproducible and significant clinical observations, rather than to suffer the inconvenience of adapting “well-established” concepts to apparently mutually exclusive observations. As a matter of fact, there exists a general resistance toward positive action under any circumstances. For instance only in recent years has there been universal recognition of the life-saving role of pressor drugs in significantly reducing the heretofore prohibitive mortality in patients with acute myocardial infarction complicated by severe hypotension.[2] The natural reluctance of the physician to transfer laboratory results to the patient has reached its greatest peak of development in the field of coronary heart disease.

Obvious clinical benefit associated with procedures which have adequate laboratory substantiation should not be categorically rejected merely because human coronary heart disease cannot be reproduced exactly in the laboratory, or because its unpredictability defies simple statistical evaluation. Indeed, if need be, it is possible to achieve “progress without statistics.”[3]

Even now, during the observation of the Tercentenary of William Harvey' death, many of the significant contributions made since his time are still engulfed in controversy. Furthermore, unless there is adequate reappraisal of certain outmoded concepts, such controversy will always be encouraged by seemingly paradoxical but impressive clinical benefits associated with such dissimiliar agents as nitroglycerin and surgical operation for coronary heart disease. Neither of these presume to produce tremendous alterations in total coronary inflow, but their admittedly limited contributions are critically decisive in the precariously compensated circulation of a potentially ischemic area.

HYDRAULIC CONSIDERATIONS

In a hydraulic system at dynamic equilibrium, the various factors which influence flow can be defined on the basis of simple hydraulic principles

(Poiseuille's law); however, with respect to pulsatile flow in the arteries, these principles should only be applied in rough estimation of these interrelations.[4-7] In the coronary circulation this application is further modified by the influence of extravascular compression. However, for purposes of this presentation, certain generalizations may be derived:

1. Blood flow is proportional to the pressure gradient in a system. Actually, as will be pointed out later, this relationship applies only within certain limits.

2. Flow is also proportional to the square of the cross-sectional area of the vessel, or to the diameter to the fourth power; at critical levels, relatively small changes in the diameter of a vessel may result in profound alterations in blood flow. In hydraulic model experiments, designed to simulate certain aspects of the coronary arterial circulation and using whole blood at body temperature (with no "peripheral resistance"), one-inch segments of hypodermic needles were inserted at the end of the system to produce various degrees of "coronary artery stenosis." Blood flow was measured with simulated "normal" pressure gradients. Average measurements of blood flow are given in Table I merely to indicate semiquantitatively these flow-pressure relationships.

TABLE I. ESTIMATED BLOOD FLOW THROUGH SIMULATED "CORONARY ARTERY" NARROWING

NEEDLE	INTERNAL DIAMETER (MM.)	BLOOD FLOW (VALUES IN DOGS)	
No. 22	0.72	4 c.c./min.	(normal backflow)
No. 20	0.90	8.5 c.c./min.	(backflow after Beck I operation)
No. 18	1.2	34	(circumflex inflow)
No. 15	1.5	100	(total coronary inflow)

3. Blood flow is inversely proportional to the viscosity. Blood with a normal hematocrit has approximately five times the viscous resistance to flow as does water.[8] An increase in hematocrit from 50 per cent to 60 per cent (such as may be associated with polycythemia, dehydration, cold, and emotional states) significantly increases relative viscosity (from 4.5 to 6.5).[9] In a critically compensated coronary circulation, such an increase in resistance to flow may be disastrous.

MEASUREMENTS OF THE CORONARY CIRCULATION

Direct measurement of coronary arterial flow can be carried out in open-chest dogs by means of a rotameter[10] and in closed-chest dogs by means of the nitrous oxide method with coronary sinus catheterization.[11-17] Although direct measurement of flow in man is obviously impractical, the nitrous oxide method with coronary sinus catheterization has provided generally reliable data, although certain considerations, especially in patients with coronary heart disease, may introduce significant errors.[18, 19] In patients undergoing heart surgery, simultaneous sampling from the coronary sinus, pulmonary artery (mixed venous blood), and systemic artery has provided an opportunity to determine oxygen (and other substrate) extraction by the heart, and an index of the adequacy of coronary arterial flow.[20]

In the laboratory,[21] the Mautz-Gregg backflow method, which measures the retrograde flow from the distal segment of an occluded coronary artery, is considered to provide a true measure of interarterial anastomotic function. These retrograde flows are a measure of the collateral bed and serve as a most valuable method for the evaluation of the efficacy of various methods designed to improve collateral circulation. For instance, in a series of 65 control dogs, the average backflow was found to be 3.8 c.c. per minute. In a series of 41 dogs previously operated (Beck I), the average backflow was found to be 8.5 c.c. per minute.

In dogs, the heart weight is estimated to be 0.9 per cent of body weight; so that, in a 15 Kg. dog, the heart weight would be approximately 135 Gm. Coronary arterial flow, which is approximately 5 per cent of the cardiac output, averages 70 c.c. per 100 Gm. of heart weight per minute.[22] Thus, in the 15 Kg. dog, total coronary flow is approximately 100 c.c. per minute. Since the circumflex artery area normally would receive approximately 34 c.c. per minute, the average backflow of approximately 4 c.c. per minute, is 11 per cent of normal inflow into this area. The average backflow of 8.5 c.c. per minute provided by operation is approximately 25 per cent of the normal inflow.

In man, average coronary inflow at rest is approximately 80 c.c. per 100 Gm. left ventricle per minute (approximately 5 per cent of the cardiac output), average oxygen consumption is 9.4 c.c. per 100 Gm. per minute, and the average oxygen extraction is 12 volumes per 100 c.c. of blood flow.[11-13] In an average man, with a cardiac output of 5 L. per minute and a heart weight of 300 Gm., total coronary flow would be approximately 250 c.c. per minute. For purposes of this presentation, let us consider a potentially ischemic circumflex coronary arterial bed with a normal resting flow of 100 c.c. per minute. In this consideration it should be stressed that, generally speaking, the clinical manifestations of coronary heart disease do not result so much from a uniform total reduction in coronary flow as from differences in oxygenation in contiguous areas.[23] As will be amply demonstrated subsequently, there is no obligate relationship between the clinical status of the patient and the total coronary inflow at rest. With a normal total inflow, incapacity and death may occur; on the other hand, in a patient with significant reduction in total inflow, symptoms may be slight or absent.

GRAPHIC REPRESENTATION OF THE CORONARY CIRCULATION

The illustrations herein presented are in the form of stylized graphs, designed to demonstrate flow-pressure relationships in the coronary circulation, with special reference to significant pathophysiologic influences. It should be emphasized that these graphs are obviously oversimplified and are, at best, only semiquantitative. Little consideration has been given to such admittedly significant factors as extravascular compression of the coronary vessels associated with systolic contraction of the heart.[22, 24] In the following part of this presentation, the section number corresponds to the similiarly numbered figure. The detailed description associated with each figure is given in the text.

1. *Normal—At Rest.*—The coronary circulation may be characterized as a high-resistance circuit,[24] in which, merely by an appropriate reduction in vascular resistance, large increases in blood flow to the myocardium are achieved. In a sense, the major coronary arteries serve as relatively passive, wide-bore tubes with a cross-sectional area normally capable of accommodating tremendous increases in blood flow (five to ten times control levels) without significant increases in perfusion (aortic) pressure.[25] For the most part, the sum total of peripheral resistance is normally determined at the level of the arterioles and small terminal arteries[24]; as mediated by metabolic, hormonal, and nervous influences, changes in the smooth muscle of these vessels regulate the bore of the coronary bed.

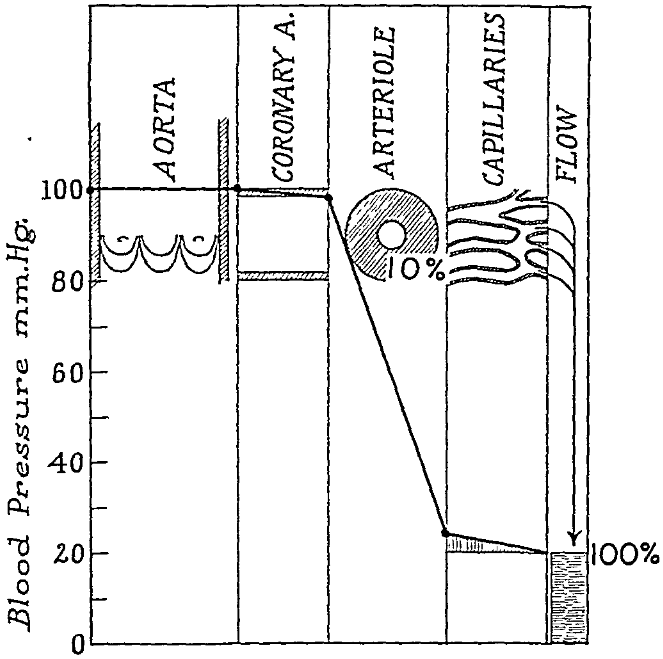

Fig. 1.—Normal—at rest. Stylized graph demonstrating flow-pressure relationships in a coronary bed. Detailed description in text. This and succeeding figures follow the same general motif. The pressure head (in mm. Hg) in each component part of the circulation is represented by the heavy line. In the interest of simplicity, the capillary pressure is assumed to average 20 mm. Hg.[24]

In Fig. 1, mean aortic perfusion pressure is shown on the graph to be 100 mm. Hg (first column). Perfusion of normal coronary arteries is accomplished without loss of pressure head (second column); the cross-sectional area of the major coronary arteries is not a determinant of flow at this level. It is primarily at the arteriolar and terminal arterial level (third column) that coronary blood flow is normally determined. Regulation of flow through the relatively passive capillary bed obtains in the form of an abrupt reduction of the pressure head at the precapillary level. A comparatively small pressure gradient across the capillary bed (fourth column) provides normal flow (fifth column).

2. *Normal—Increased Flow.*—Normally, at rest, myocardial oxygen extraction is so high that increased oxygen consumption by the heart, as during exercise, is achieved primarily by increased flow.[26] This is readily accomplished

by a decrease in arteriolar resistance so that the pressure head arrives at the capillary bed at a higher level. A twofold increase in the arteriolar bed results in a fourfold increase in flow with no significant alteration in perfusion pressure or in major coronary artery cross-sectional area. At extremes of physical exercise or when occlusive disease interferes with coronary flow, an increase in oxygen extraction provides an important compensatory mechanism.[27] (Fig. 2.)

3. *Severe Coronary Stenosis.*—With progressive reduction of the lumen of a major coronary artery, coronary flow at rest is not compromised until the cross-sectional area approaches approximately 10 per cent of normal. At this level, the artery bore also becomes a determinant of coronary flow and reduces the pressure head arriving at the arteriolar bed so that, unless there is a reduction in arteriolar resistance, a deficit in coronary flow results, even at rest.

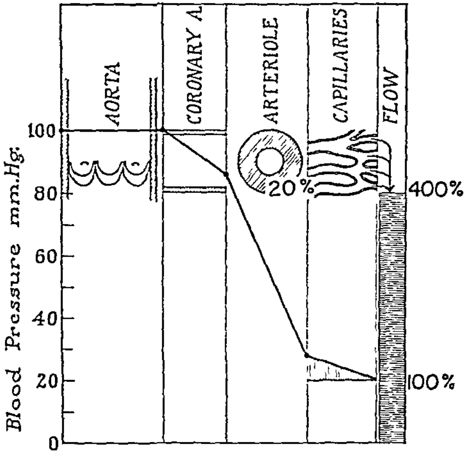

Fig. 2.—Normal—increased flow.

During exercise, in which increased flow is needed, an even greater deficit occurs. In cardiac hypertrophy wherein coronary flow per 100 Gm. of heart muscle is normal,[11] total coronary flow at rest is increased. A reduction in cross-sectional area, which would still allow adequate flow in the normal, causes a deficit in flow in the presence of hypertrophy. (Fig. 3.)

4. *Severe Coronary Stenosis—Compensatory Dilatation.*—At this critical level, the cross-sectional area of the major coronary artery and the arteriolar bed are both determinants of coronary flow; compensatory arteriolar dilatation is still effective in restoring flow to normal at rest (although coronary reserve is obviously reduced). The distribution of coronary blood flow is determined by local resistance. Hypoxia is a powerful coronary vasodilator[27-30]; as a given area

becomes ischemic, its resistance falls, thus encouraging preferential flow to that area. Parenthetically, this reduction in resistance, which is a relative increase in pressure gradients from adjacent beds, also encourages intercoronary collateral blood flow from these regions. (Fig. 4.)

5. *Severe Coronary Stenosis—Exercise.*—Even at this critical level, total coronary flow can still be raised above 100 per cent of normal by maximal

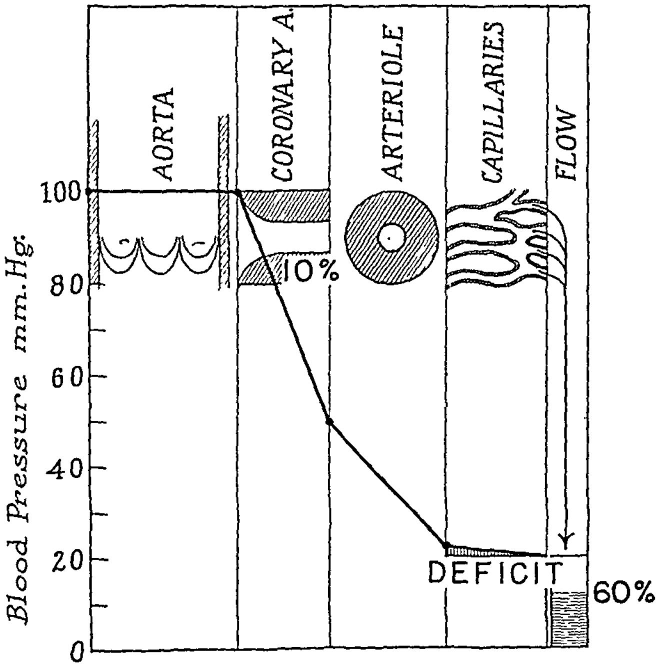

Fig. 3.—Severe coronary stenosis.

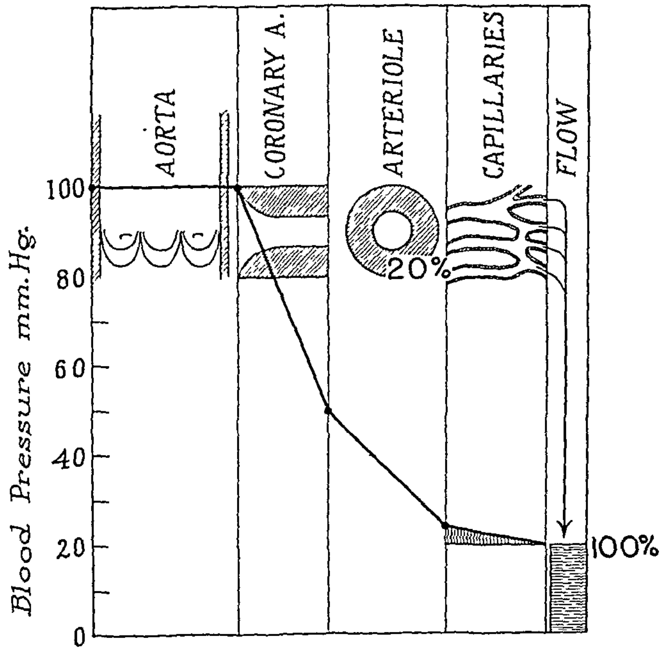

Fig. 4.—Severe coronary stenosis—compensatory anteriolar dilatation.

arteriolar dilatation. Nitroglycerin (as is the case with other true coronary vasodilators) can be shown to exert its effect at the arteriolar level; presumably, its clinical benefit depends upon potentiation of the vasodilatation produced by local ischemia.

However, at those levels of flow in which the arterial stenosis becomes the sole determinant of coronary flow, no further increase in flow is possible despite a maximal reduction in resistance. (Fig. 5.)

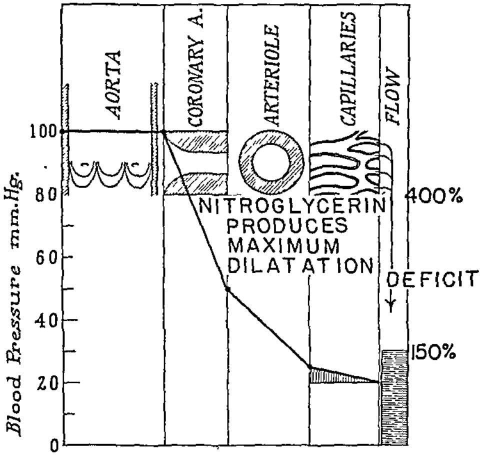

Fig 5.—Severe coronary stenosis—exercise (400 per cent required).

6. *Very Severe Stenosis—Maximum Arteriolar Dilatation.*—With a slight further reduction in lumen (down to approximately 5 per cent of its cross-sectional area), the major artery now becomes the sole determinant of coronary flow so that, despite maximum arteriolar dilatation, a deficit occurs. Clinically, this marks the onset of (1) status anginosus, (2) sudden death associated with oxygen differentials between the ischemic area and adjacent well-oxygenated areas,[23] and (3) acute myocardial infarction, even without complete artery occlusion. (Fig. 6.)

7. *Very Severe Stenosis—Small Intercoronary.*—With reduction of cross-sectional area below a critical level (even though complete occlusion has not yet occurred), catastrophe is imminent unless adequate development of functional collaterals has preceded the occlusive process. If this has occurred, a potential deficit to a given area may be averted. With an adequate pressure gradient, a group of "small" collateral vessels (however produced) may provide a comparatively abundant blood supply to a potentially ischemic area. For instance, given a large collateral vessel, or a group of vessels, with an aggregate diameter

of 2 mm. and a pressure gradient from the "donor" artery to the "recipient" capillary bed of 80 mm. Hg (100 versus 20 mm. Hg), a blood flow of 50 to 100 c.c. per minute may be achieved, which may well approach the previous normal resting inflow to that area. (Fig. 7.)

8. *Severe Coronary Stenosis—Hypertension.*—In a fundamental sense, the primary determinant of coronary blood flow is the perfusion pressure existing

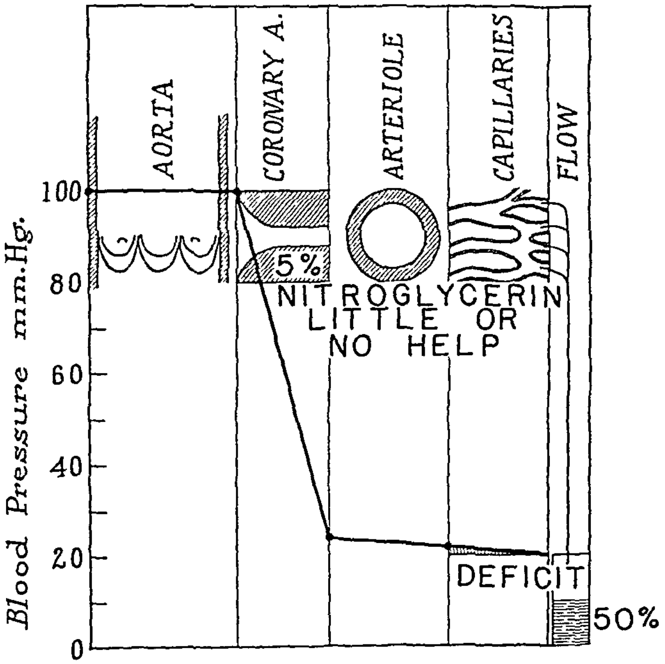

Fig. 6.—Very severe stenosis—maximum arteriolar dilatation.

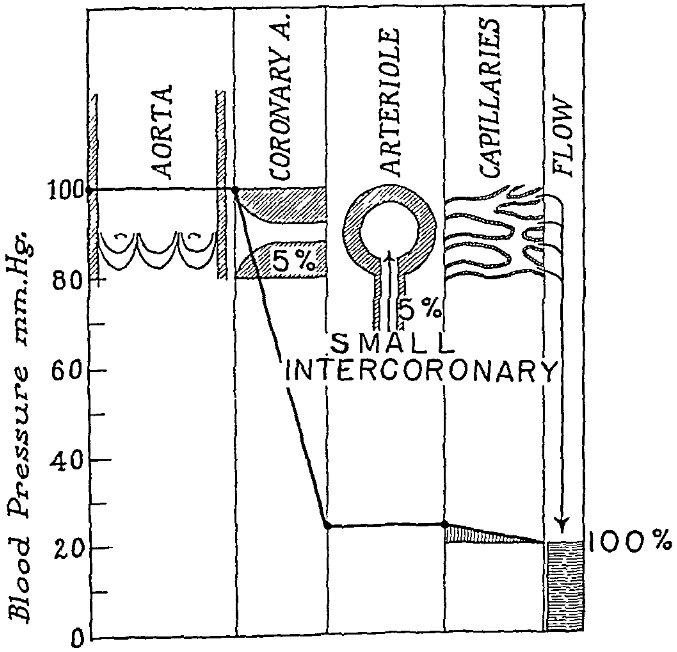

Fig. 7.—Very severe stenosis—small intercoronary.

at the coronary ostia and, more important, the pressure gradient which ultimately drives the blood across the capillary bed. Hypertension, per se, increases the work of the heart and, particularly when cardiac hypertrophy has occurred, myocardial oxygen utilization and coronary blood flow requirements may be many times the normal.[11] Myocardial ischemia may then result from a degree of coronary stenosis which normally would have permitted adequate flow.

When there is severe coronary artery stenosis, an all-or-none relationship *may obtain; with normal* aortic perfusion pressure, there is no blood flow to a given area, whereas when hypertensive levels are reached, only then, is there achievement of significant blood flow. Regardless of the increased heart work, attainment of an adequate pressure head becomes the most overriding consideration.

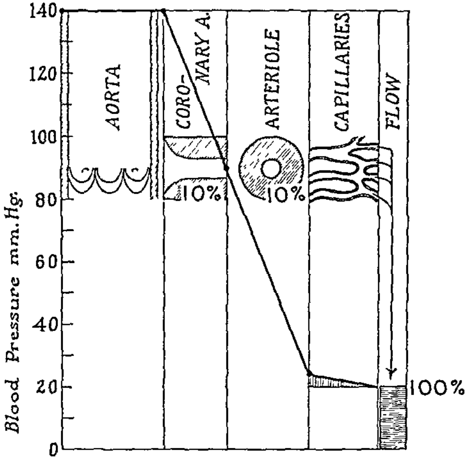

Fig. 8.—Severe coronary stenosis—hypertension.

Fig. 8 is somewhat idealized to emphasize the hemodynamic benefits of increased perfusion pressure. Obviously, the increase in heart work necessary to raise aortic pressure also increases the metabolic needs of the heart. However, in a critically compensated system, the increased flow and increased oxygen extraction provided results in an over-all salutory effect.

9. *Severe Coronary Stenosis—Hypotension.*—Conversely, when reduced below a critical level, perfusion pressure becomes inadequate to overcome viscous resistance to flow and a deleterious relationship is established. Coronary stenosis may produce such a disadvantageous situation at otherwise insignificant reductions in pressure.

Although there is some degree of obligate relationship between blood flow and the pressure gradient, under certain conditions simple hydraulic flow-pressure correlations do not obtain. For instance, for a given pressure gradient, the variability of vascular distensibility at different perfusion levels may significantly alter blood flow.[31] Normally, viscous resistance is such[5] that the initiation of flow through small vessels requires a pressure gradient of at least 10 mm. Hg (yield pressure). Even in the absence of significant coronary disease, interaction of various factors may increase viscous resistance to flow to such a degree that a given area of myocardium may be rendered ischemic. (Fig. 9.)

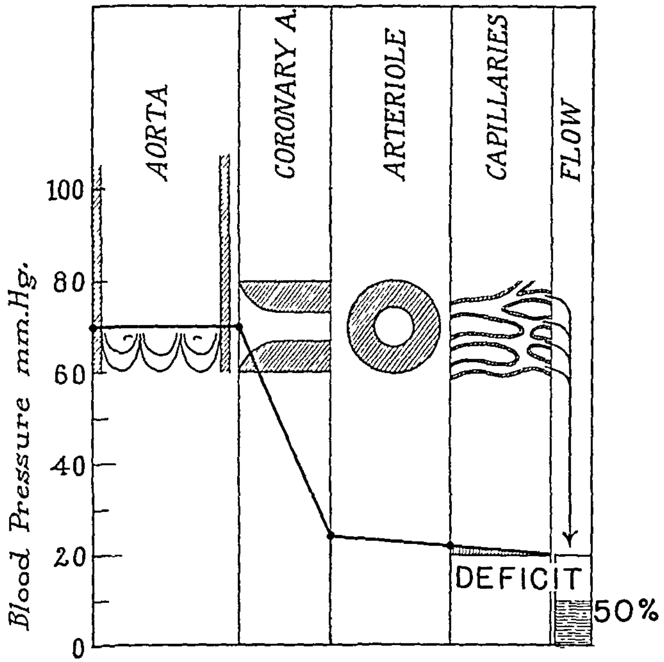

Fig. 9.—Severe coronary stenosis—hypotension.

Clinically, an apparently paradoxical response to antihypertensive therapy has been observed quite frequently.[32, 33] Instead of the anticipated alleviation of symptoms, in patients with hypertension, reduction of blood pressure to "normal" has precipitated symptoms of coronary insufficiency in many patients, with occasional catastrophic consequences. Obviously, in patients with significant coronary artery disease, loss of perfusion pressure in a critically compensated coronary circulation is disadvantageous, even though heart work is decreased. Thus, the medical reduction of aortic pressure is not necessarily desirable.

As previously mentioned, although the deleterious consequences of severe hypotension associated with acute myocardial infarction have long been recognized, the benefit of pressor drugs may still be considered questionable. In producing peripheral vasoconstriction, associated with a relative reduction in coronary resistance,[34] improved perfusion of patent coronary vessels is achieved; previously inadequate vessels may now maintain viability of peripheral regions of the infarcted area in anticipation of the eventual restoration of contractility.

Worthy of consideration as an extreme but not altogether fantastic extension of these considerations, in acute myocardial infarction, elevation of blood pressure to hypertensive levels may actually be of value in reducing the degree of muscle damage

VASCULAR REACTIVITY

The coronary vascular bed exhibits a remarkable degree of reactivity in response to various stimulants[3] Hypoxia or, more specifically, myocardial ischemia is considered to be the primary physiologic regulator of such reactivity[27, 30, 36, 39] Even in the presence of severe coronary artery sclerosis, such reactivity is still a major determinant of coronary blood flow

Many patients with coronary heart disease give a most impressive demonstration of the reactivity of this vascular bed Soon after arising in the morning, even slight effort results in anginal pain, and necessitates the use of nitroglycerin As the day progresses, however, with an actual increase in economically productive activity, there is a real reduction in pain so that by evening, unless a large meal is consumed to put an extra load on the circulation, there is less or no discomfort associated with a greater degree of activity than that which produced severe distress earlier A similar phenomenon is frequently reported from the golf course At the start, for the first few holes, the patient suffers frequent angina but, as the game progresses, a "second wind" effect supervenes, and the latter part of the game is played with little or no symptoms This "second wind" phenomenon is related to adaptation and progressive vasodilatation distal to a stenotic area and represents a significant observation of profound practical importance

In response to various hemodynamic and pharmacologic influences, coronary vasodilatation is quite salutary in some but detrimental in others[10, 47] As has been so well characterized by Gregg and Sabiston,[27] when increased coronary flow is achieved without increased myocardial metabolism, a state of "benign" vasodilatation obtains, however, if increased cardiac metabolism accompanies the increase in flow, this is aptly termed "malignant" dilatation

In the presence of severe coronary artery stenosis, the adequacy of blood flow to a potentially ischemic area, which is in a state of precarious compensation, depends upon "active" sustained maximal arteriolar dilatation, which in turn is activated by local hypoxia Any factor (reflex, or otherwise), which even slightly reduces this dilatation, may be catastrophic, despite the fact that vascular resistance may still be much lower than normal

Despite the remarkable clinical relief afforded by nitroglycerin, its mode of benefit and, indeed, its very respectability have long been suspect, presumably these doubts derive from the misapprehension that dilatation of rigid diseased coronary arteries must obtain in order to increase coronary blood flow

In the laboratory, such "benign" vasodilators as nitroglycerin can be shown to increase coronary flow, with blood pressure and cardiac output kept constant[48, 53] These vasodilators, per se, appear to have no immediate primary effect on collateral blood flow[54] Although other specific chemical or metabolic influences of these agents have been implicated,[55] and presumably disproved,[48, 56] this activity can be logically defined on a purely hemodynamic basis. Incidentally, the remarkable clinical benefit derived from nitroglycerin is further evidence that relatively slight alterations in coronary flow can have profound consequences.

Although there may well be valid objection to its use under special conditions,[57, 58] generally there is little doubt as to its "benign" specificity.[59] By way of "objective" evidence in man, electrocardiographic and ballistocardiograph abnormalities due to coronary insufficiency may be "normalized" by the administration of nitroglycerin.[57-59] A priori, the clinical benefit derived from nitroglycerin is an expression of the potentiation of myocardial ischemia plus nitroglycerin in achieving maximal vasodilatation.

Theoretical arguments to the contrary notwithstanding, prophylactic administration of nitroglycerin actually prevents pain associated with a given amount of activity (by "benign" vasodilatation). Long-term administration of such vasodilators may actually aid in the development of collaterals[60]; however, due consideration is necessary before such may be applied to man.

Blood vessels in different vascular beds react differently under the same circumstances. For instance, in hemorrhagic hypotension, coronary resistance decreases, so that normal coronary flow is maintained; whereas peripheral circulation is decreased.[61] Certainly, vasospastic phenomena are well recognized in peripheral vascular disease,[35, 62] and there is little reason to doubt that such phenomena may well play an important role in the clinical manifestations of coronary heart disease.

Since most coronary catastrophes occur at "rest," it has been assumed that undue stress is rarely the cause, except in certain situations of most strenuous exertion.[63] Contrary to most published reports, in our own history-taking, we have been profoundly impressed by an unexpectedly frequent cause and effect relationship between severe stress and subsequent "heart attacks." which attacks mostly occurred at rest, but the proximate cause appeared undeniable. The fact that there is a period of relative inactivity between the stress and the actual attack should not be allowed to obscure their relationship.

A careful retrospective evaluation of our studies and other published reports[28, 49] reveals a heretofore overlooked, but potentially significant observation: The increase in coronary blood flow, associated with various forms of coronary vasodilator activity, is almost invariably followed by a "rebound," or "dip," phenomenon,[28, 49] so that coronary blood flow is then below normal for extended periods of time. It is merely suggested that such a period of relative vasoconstriction following a period of increased blood flow may well have obvious clinical implications.

It is not altogether fantastic to postulate the following sequence of events: As a given coronary artery progressively narrows, increasing arteriolar dilatation in response to local hypoxia compensates to maintain normal flow to that area. However, eventually, despite maximal dilatation, artery stensis becomes so severe as to reduce inflow into this area, so that the myocardium becomes ischemic, and profound alterations in cellular metabolism occur. Break-down products of ischemia, liberated via the now permeable cell wall either reflexly or by direct stimulation, produce profound vasoconstriction of the hitherto excessively dilated arterioles, with resultant stasis of blood in the diseased artery. Thus, there is complete obstruction of flow to a given area of myocardium with subsequent necrosis and scar formation. Furthermore, as a result of the sudden development of stasis in the artery, and as an almost incidental secondary phenomenon, thrombosis may occur in that artery segment which is most vulnerable. In other words, that event, toward which we have directed most of our attention as the primary cause of heart attacks, fantastic as it may seem, may conceivably be an incidental by-product of another primary cause.

In this regard, a most intriguing inference is suggested by a series of reports made by European observers.[64, 65] These are concerned with the occurrence of sudden death in at least 50 presumably healthy males engaged in the manufacture of explosives. In each case, there was prolonged continuous exposure to nitroglycol, a volatile nitrite, whose mode of action in man is probably identical with that of nitroglycerin and certain other clinically useful vasodilators.[66] Routine physical examinations a short time prior to these deaths had revealed no significant abnormalities; there was a tendency to bradycardia and slight hypotension; there were no unusual symptoms.

Invariably, sudden death occurred during an unusually prolonged period away from work (vacation or long week end), and appeared to be associated with some degree of mild to marked exertion. The sudden collapse was preceded by precordial distress and "weakness" in some cases Although it is presumed that death was caused by some form of coronary insufficiency, post-mortem examination revealed essentially normal coronary arteries and myocardium.

These observations have given rise to much speculation,[57, 64-66] none of which appears to account satisfactorily for these sudden deaths. As a logical extension of the concepts herein presented, this intriguing sequence of events lends itself to the following tantalizing explanation: Prolonged exposure to a volatile and potent coronary vasodilator (nitroglycol) had produced in these men a sustained "active" coronary arteriolar dilatation, in which situation the normal physiologic stimuli (hypoxia) were relatively ineffective. Thus, the regulation of vascular resistance was impaired by this "artificial" influence, and had indeed become dependent upon it. When this artificial vasodilator environment was withdrawn, the balance of influences controlling vascular reactivity was disrupted and a "rebound" of relative vasoconstriction ensued. When, in response to the subject's increased activity, normal stimuli to provide the necessary increase in coronary flow were ineffective, a sudden marked deficit occurred, with complete disruption of all myocardial compensatory mechanisms. As a terminal event, arteriolar vasoconstriction may have resulted from this "rebound" phenomenon.

This explanation is not altogether fantastic; similar disturbances in regulation of vital functions abound in clinical states. In pulmonary conditions in which there is alveolar hypoventilation, chronic carbon dioxide retention results in a diminished respiratory response to carbon dioxide. Anoxia, operating via carotid body chemoreceptors, becomes the primary stimulant to breathing. In such patients, the administration of high concentrations of oxygen, with a rise in arterial oxygen tension, results in reduced ventilation, further carbon dioxide and bicarbonate retention, narcosis, and death.

INTERARTERIAL COMMUNICATIONS

Basically, the extent of coronary arterial occlusion, per se, is not the primary determinant of the clinical manifestations of coronary heart disease; rather, it is the adequacy of the collateral circulation which determines the clinical course.[63, 67-73] Although various other circulatory channels, intra- and extracardiac in origin, have been implicated,[74-80] by and large, the collateral circulation of the myocardium is almost exclusively a function of interarterial anastomoses.

In a fundamental sense, coronary heart disease is a grim tug-of-war, or battle for survival, between the relentless occlusive process on the one hand and a compensating collateral circulation on the other. The patient who develops clinical evidence of coronary heart disease has arrived at this stage of precarious compensation.

Under certain optimum conditions, this delicate balance may be so well preserved that the patient's clinical condition becomes "stable." Generally, this "stability" should not be considered to indicate that, by some miraculous means, the occlusive process has suddenly been halted, but only that it is progressively "neutralized." Thus, the patient is able to maintain economic productivity and well-being almost indefinitely, unless some relatively "insignificant" factor, if only momentarily, upsets this delicate balance and causes death. By the same token, death may be averted by just a little "extra" protection in the right place. Ultimately, with a marked reduction in total coronary inflow, however well distributed, myocardial failure ensues.

Although the excellent studies of Gross,[81] and of Spalteholz,[82] had long ago demonstrated the anatomic features of intercoronary arterial communications, the criterially decisive role of the collateral circulation in protecting the myocardium was not established until the truly momentous work by Blumgart, Schlesinger, and co-workers.[70-73] These studies thus helped dispel the mystery surrounding the inexplicable lack of obligate correlation between the occlusive process in the coronary arteries and the myocardial response thereto.

This monumental work should serve as the logical basis for the therapeutic attack on coronary heart disease; as indeed was well stated in 1951, "Any regimen that would produce interarterial anastomosis so that the coronary arteries were no longer functionally end-arteries would mitigate the clinical manifestations of coronary artery disease. Knowledge of the factors influencing the development of interarterial coronary anastomosis, therefore, would be of great practical value."[72] Unfortunately however, the logical application of these observations in the definitive treatment of coronary heart disease has been obfuscated by a remarkable series of non sequiturs.[83]

In hearts with chronic occlusion of one or even, apparently, of all major coronary arteries, the absence of significant myocardial injury should no longer be considered a medical curiosity which defies scientific explanation, but rather an impressive demonstration of the tremendous potential for protecting the myocardium provided by adequate intercoronary collaterals. With adequate collaterals to provide uniform distribution via a final common pathway for the ubiquitous red blood cell, the integrity of the myocardium may be maintained almost indefinitely, despite multiple complete coronary artery occlusions.

The activation of collaterals, in response to adequate pressure gradients in adjacent coronary vascular beds, is certainly no more remarkable than many other natural and specific reactions, such as antibody production against a specific infectious agent. The absence of such collaterals[72] in hearts without chronic, complete (or almost complete) coronary artery occlusion is merely an expression of the fact that coronary flow (at rest) to a given area is not compromised until the cross sectional area is reduced to a small fraction of normal. In response to a given stimulus, there is great variation in man in his ability to develop collaterals. As occurs in other clinical conditions (for instance, susceptibility to carcinoma), this may well be a function of hereditary predisposition. The well-recognized severe disposition, in some families, to a particularly malignant form of coronary heart disease, may actually be a hereditary indisposition to the development of collateral circulation.

Generally, any factor which decreases arteriolar resistance (thereby increasing coronary blood flow), and thus exaggerates pressure gradients from adjacent pre-arteriolar beds, will stimulate increased collateral flow. Such appears to be the case with known stimulators of collateral circulation: anemia[8, 72] anoxia,[72] exercise,[25] and such pharmacologic agents as the nitrites.[60]

Of profound significance is the fact that such procedures as the Beck I operation increase collateral circulation, even in the absence of significant pressure gradients.[84-90] Should there be acute arterial occlusion, such increased collateral circulation, provided in advance, even though it be insufficient to maintain contractility, will maintain viability of the myocardium The eventual increase in collateral flow to this low pressure bed then restores contractility so that there is no destruction of heart muscle.

There can be little doubt that chronic coronary artery stenosis, with resulting myocardial ischemia, is the stimulus, par excellence, in the activitation of intercoronary arterial communications.[70-73] However, in view of the exorbitant mortality associated with this disease, the assumption that patients with clinical manifestations of coronary heart disease (and myocardial hypoxia) are diligently developing adequate "natural" collaterals is indeed unrealistic. Inherent in such an assumption is the implication that the need for further definitive therapy is obviated. This may perhaps be likened to the following nonsequitor· In a patient with an overwhelming infection, since a specific immune reaction will produce high antibody titers, there is no need for antbiotic therapy.

In view of the remarkable protection provided by a truly adequate collateral circulation, the post-mortem demonstration of "adequate" collaterals in a patient who is dead as a result of coronary heart disease (despite the presence of such collaterals which are presumably more than adequate to prevent death) is apparently paradoxical

In the presence of truly adequate collaterals, a most remarkable degree of protection is afforded in man.[70, 72, 91] When controlled graded coronary occlusion is carried out in dogs, the development of an adequate collateral circulation enables the animal to survive total occlusion, and may indeed prevent myocardial infarction; in which case there can be no question regarding the far-reaching benefits of a naturally stimulated collateral circulation.[92, 93] Thus, the establishment of adequate intercoronaries, however produced, *in advance* of total occlusion, is truly protective; and any procedure which enhances the establishment of this collateral circulation will provide such far-reaching benefit that this will defy comprehension based upon "established old concepts," in view of the admittedly small amounts of blood made available to potentially ischemic areas.

Although we originally presumed we might help in resolving controversy by means of a more or less unified concept of coronary heart disease, re-evaluation of the role of the collateral circulation has inevitably led us into a new and wholly unexpected development, which might well be considered the last straw in this patently iconoclastic presentation. Heretofore, in the face of all the other raging controversies, the only "sacred cow" in coronary heart disease has been the universally acclaimed collateral circulation. As with the "benign" and "malignant" forms of coronary vascular dilatation, so we find, the collateral circulation itself may well be "benign" or "malignant"!

Adequate intercoronary anastomoses in advance of the occlusive process are truly beneficial; the extensive collaterals resulting from coronary occlusion and myocardial infarction are not necessarily salutary and may actually be deleterious. In encouraging perfusion of a scar, which represents a low resistance bed, a functional arteriovenous fistula may be produced. Preferential flow to this area would then deprive adjacent and still viable myocardium of critically needed blood.

In direct studies during coronary heart surgery, we have found myocardial oxygen extraction (artery-coronary sinus oxygen difference) to be within normal limits, even in patients with severe symptoms of coronary heart disease; however, in those patients with a significant degree of myocardial scarring as a consequence of massive myocardial infarction, the oxygen content in the coronary sinus blood is frequently found to be significantly elevated, indicating a reduction in myocardial oxygen extraction.[20] This apparent paradox is interpreted as further evidence that, in an already compromised coronary circulation, significant amounts of critically needed blood are perfusing the heart without oxygenating the myocardium. Such a phenomenon has been observed by others,[11, 19] but the deleterious significance may have escaped detection.

Clinically, a seemingly inexplicable course of events may well be associated with this concept of malignant collaterals: Following acute myocardial infarction, there is apparent clinical improvement until the tenth to fourteenth day, at which time, rapid deterioration and death supervene. At post-mortem examination, there is no "new" cause for sudden death; the healing infarct and old occlusion, at this stage, are per se, considered as "inadequate" to account for such circulatory deterioration. It is perhaps more than coincidental that chronologic relations are such that the full development of collateral circulation is generally achieved by this time.[73, 94-98] In patients who survive acute myocardial infarction, "recovery" is marked by gradual circulatory deterioration, with little or no anginal pain, but characterized by easy fatigability, weakness, shortness of breath, tachycardia, reduced pulse pressure, and a relentless downhill course, leading inevitably to death from congestive heart failure. Again, the post-mortem findings are generally considered "inadequate," even by the pathologist, in elucidating this course of events. The amount of heart muscle damage, per se, is inconsistent with the catastrophic clinical consequences. Furthermore, when injection and dissection studies are performed, the collateral circulation is "more than adequate" to prevent death.[70, 97]

If this pernicious influence of postinfarction collaterals (not to be confused with "benign" and remarkably protective preinfarction collaterals) can be adequately substantiated, in addition to the obvious implications of such a profound alteration in a "basic" concept of cardiology, in such patients we will also have the first really valid indication for surgical excision of a myocardial scar or, perhaps, if technically feasible, just ligation of the "malignant" collaterals.

SURGICAL TREATMENT FOR CORONARY HEART DISEASE: DIRECT VS INDIRECT

It is not our intention in this publication to discuss, per se, surgical treatment; however, suffice it to say that, in our desire to achieve the "direct" surgical approach to this disease, we must not disregard certain fundamental (but neglected) considerations with regard to the pathophysiology of coronary heart disease. Such prodigious technical achievements as coronary endarterectomy[99] and anastomotic bypass of arterial occlusion almost pale into insignificance as compared with the other formidable and deceptive pitfalls inherent in this "direct" approach. Presumably, contrast visualization of the coronary arteries in man will eventually be feasible (if somewhat dangerous); thus, in a patient with clinical symptoms of coronary heart disease, "localization" of obstructions in the coronary circulation will allow the surgeon to select at least certain diseased segments for the "direct" approach. (It is unrealistic to anticipate the application of such procedures to more than one, or perhaps two, separate obstructions in a given patient.)

It is axiomatic that there is no obligate relationship between the extent and location of the occlusive process in the coronary arteries and the clinical consequences thereof.[70] Only "moderate" stenosis of one artery may result in death; on the other hand, chronic multiple complete occlusions of two or more arteries may produce no symptoms and, indeed, no myocardial damage. In a fundamental sense, the patient's symptoms are not due to the actual occlusive process in the coronary arteries, but to the response of the myocardium. Coronary occlusion, per se, is not the clinical disease. Even consideration of endarterectomy as an "emergency" procedure is unrealistic. Although coronary thrombectomy is "like thrombectomy for arterial thrombosis elsewhere,"[99] the myocardium is unlike other organs (except, to a certain degree, the brain), in that within 20 to 30 minutes after occlusion irreversible damage has occurred.[70]

In a patient with coronary heart disease, the unwarranted assumption that the functional disturbance is due to a given obstruction of a coronary artery is most ill advised and may have disastrous consequences. Restoration of flow into a given area of myocardium may not only have no beneficial effect but, if flow is restored to a scar in a previously infarcted region, preferential perfusion of a low-resistance area may then deprive viable myocardium of blood which is already in short supply. Thus, a deleterious effect is produced, as may well have been (inadvertently) recently demonstrated.[90]

The patient with coronary heart disease has a precariously balanced circulation; adequate intercoronary collaterals may maintain the dynamic integrity of the heart almost indefinitely. Any procedure which enhances the production of collaterals in advance of the occlusive process will be remarkably effective. On the other hand, a ''direct'' approach, which may actually disrupt this balance, may have catastrophic consequences.

SUMMARY AND CONCLUSIONS

In a fundamental sense, the dynamics of coronary heart disease may be characterized by the following axioms:

1. The fate of the myocardium, and indeed the fate of the patient, depends upon the amount of blood available to a potentially ischemic area.
2. There is a complete lack of obligate correlation between the degree of occlusive disease in the coronary arteries and the clinical consequences thereof. It has been convincingly demonstrated that complete coronary artery occlusion does not necessarily result in myocardial infarction; myocardial infarction may occur without complete artery occlusion; the site of myocardial infarct is not necessarily related to the site of coronary occlusion.
3. In the presence of a truly adequate intercoronary arterial collateral system, the tremendous potential for preservation of the myocardium is preserved even when total cross-sectional area of the major coronary arteries is profoundly reduced to a mere fraction of normal.

The following can be considered as corollaries to these axioms:

1. The symptoms of coronary heart disease are not necessarily correlated with total coronary inflow.
2. Rejection of the "indirect," and application of the "direct" surgical approach to the problem of coronary heart disease, is frequently based upon the false premise that the disease process has obviously already produced maximum intercoronary communications; therefore, little can be achieved in stimulating more collateral circulation.
3. Generally, it is the uniformity of distribution rather than total inflow that determines the clinical manifestations of coronary heart disease.

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DISCUSSION

DR. BERNARD L. BROFMAN, Cleveland, Ohio (Closing).—There can be little doubt that the ultimate solution to this destructive disease lies in medical prevention of the occlusive process in the coronary arteries. Whatever the ultimate role of surgery in the treatment of coronary heart disease, one of Dr. Beck's most significant contributions in this field has been to encourage the coordinated effort of the medical cardiologist and the cardiac surgeon and in correlating experimental, hemodynamic, clinical, pathophysiologic, and bioelectrical observations and *applying* them for the benefit of the patient. Certainly, without such a comprehensive approach, one cannot achieve a series of 100 consecutive operations without operative mortality; nor can one demonstrate an almost unbelievable degree of *clinical improvement* and *increased longevity* in 9 out of 10 patients operated upon.

[The corrected discussion by Dr. Vineberg has not been returned by the discusser. —EDITOR]

ATRIOSEPTOPEXY: A SIMPLIFIED NEW TECHNIQUE

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ALTHOUGH closure of human atrial septal defects was first suggested by Cohn[1] in 1947, and was accomplished partially in 1948 by Murray[2] by anterior-posterior suturing of the atrium in the line of the septum, it was not until Jan. 11, 1952[3] that a human atrial septal defect was completely closed by one of us (C. P. B.) as shown by postoperative catheterization. The method amounted essentially to invaginating a portion of the consistently redundant or dilated right atrial wall against the septal defect, to the periphery of which it then was securely sutured under digital guidance. This method of closure was termed "atrioseptopexy." Since its innovation, a number of logical modifications have been devised and applied for the several anatomic variations which may be encountered[4-6] (Fig. 1, *A*, *B*, *C*).

Since that time, other methods have been developed and tested extensively. These include the closed method of Sondergaard in which the septal defect is constricted by a circumferentially placed tape or suture, and the semi-open "well" method of Gross[8] which has been elaborated upon by Kirklin.[9] (More recently, Gross[10] has adopted the method of atrioseptopexy for the majority of his cases.) Finally, there are the open techniques of Lewis and Taufic,[11, 12] and Swan,[13] using hypothermia, and of Gibbon,[14] DeWall,[15] and others using cardiac by-pass with a pump oxygenator.

Although these latter techniques in time may prove to be applicable and safe for this purpose, their very considerable presently existing hazards have led us to restrict their use to the more complicated intracardiac lesions which are not remediable by closed methods. Atrioseptopexy, in our hands, has proved both safe and practicable, and has been employed routinely by us with good results in properly selected cases of ostium secundum defect (Table I). We have had poor results in patients with ostium primum lesions, and prefer to operate upon these by an open technique using a pump oxygenator.[16] Also, we have had poor results in patients in whom the shunt has become either

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reversed or balanced,[6] apparently because of the extreme pulmonary vascular change which has taken place. We feel that these patients have become irretrievable by any surgical means.

TABLE I. RESULTS OF ATRIOSEPTOPEXY IN OSTIUM SECUNDUM PATIENTS WITH A LEFT-TO-RIGHT SHUNT OF MORE THAN 1.5 LITERS PER MINUTE

NUMBER OF CASES	NUMBER OF DEATHS	PERCENTAGE MORTALITY	POSTOPERATIVE CATHETERIZATION	NUMBER CLOSED	PERCENTAGE OF CLOSURE
74	4	5	40	38	95

Because of the low mortality (5 per cent) and the high number of successful closures (95 per cent by catheterization) which is obtained by atrioseptopexy, we feel that this is the method of choice at the present time.

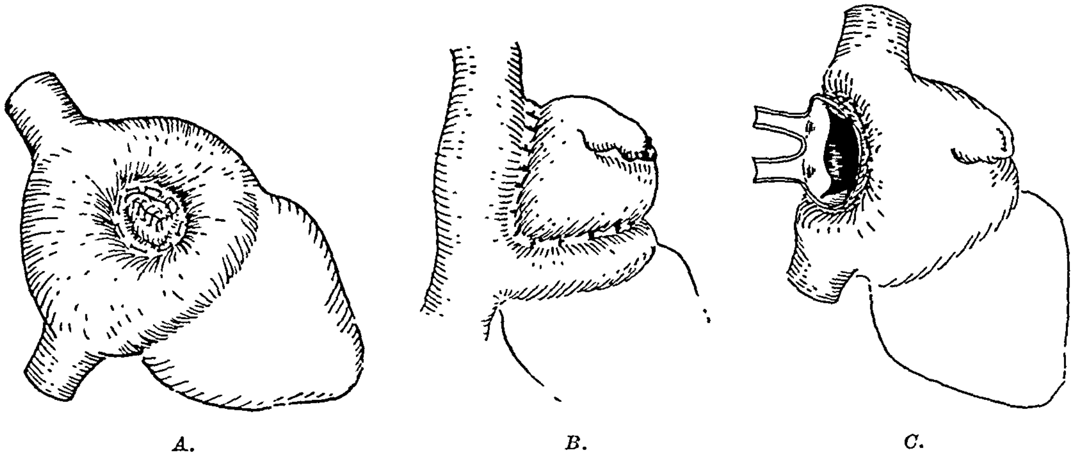

Fig. 1.—*A*, Classical atrioseptopexy. Invagination of the redundant right atrial wall and suture approximation to the periphery of the defect, suitable for moderate-sized central defects.

B, Atrioseptopexy which displaces the effective right atrial chamber posterior and caudad to the defect which remains anatomically existent.

C, Atrioseptopexy for retransposition of anomalous right pulmonary veins after enlargement of septal defect. If none exists, one may be created by the use of the cardioscope or by a bistoury knife and scissors (guided by the intracardiac finger).

However, at times, one of us (C. P. B.) has experienced considerable technical difficulty in performing this operation, particularly when the right atrial chamber is very large and the lower (caudad) portion of the atrial septal remnant lies very deep within the atrial cavity. Should this septal ridge prove to be small or unusually fragile, it may be nearly impossible to pass a properly directed needle through both the lateral atrial wall and this tissue at the same time. Tearing of the septal margin has resulted from persistence in such effort and, on one occasion, has been responsible for the persistence of a small residual interatrial communication.

For these reasons, we would like to present what we feel is a significant advance, first suggested by the senior author (T. H.), in the technique of performing atrioseptopexy. In brief, this consists of introducing the guiding left index finger into the *left* atrium from the right side of the heart, so that it lies behind or to the left of the defect, instead of introducing it through the right auricular appendage, as has been our custom previously. This method of entrance into the left atrium from the right side is the same as that which we have described for the performance of mitral commissurotomy from the right side.[17, 18] Since it is not necessary to constrict the base of the auricular appendage with a purse-string suture, a significantly greater amount of right atrial wall thereby is rendered available for the septal repair.

With the intracardiac finger tip inserted to the left of the septal remnant and with the left thumb outside of the dilated atrium, it is easy to bring into apposition the lateral septal wall and the rim of the defect, thus greatly facilitating the placement of sutures for permanent closure of the defect. Rather small suturing needles (curved) are satisfactory once these two tissue layers have been brought into neat contact.

OPERATIVE TECHNIQUE

The patient is placed upon the operating table in the supine position, and a curved submammary incision is made from the right sternal border to the mid-axilla. The fibers of the pectoralis major muscle are separated and divided as necessary, and the right fourth intercostal space is opened. The internal mammary vessels are divided individually and ligated. If necessary, the costal cartilage of the fourth rib is separated from its sternal attachment to increase the exposure.

The pericardial sac is incised longitudinally just anterior to the phrenic nerve. The lower extremity of this incision is angulated forward to the sternal margin to obtain a wide exposure of the right atrium. By manual retraction of the right atrium with a gauze pad medially and anteriorly, by an assistant's hand, the interatrial groove is exposed.

The epicardium covering this interatrial fossa is dissected longitudinal for a distance of 1.5 cm. in the line of the groove, and to a depth of approximately 1 cm. It is encircled widely by a No. 2 nylon purse-string suture placed deeply into (actually through) the respective atrial walls on each side of the groove. The suture is incorporated within a Rumel-Belmont tourniquet for flexible reliable hemostatic control. Using a swedged-on curved needle, a No. 3-0 cotton stitch is placed just beyond either extremity of the dissection in such a way as to hold the two atria together, thus obviating any tendency toward spontaneous further separation. The ends of these stay sutures are left long.

A heavy (No. 2) braided nylon suture with a swedged-on curved needle is passed and repassed through the everted lips of the epicardial incision in such a way as to ensure later eversion of the to-be-incised septal aspect of the left atrial wall after the intracardiac manipulations through this entry portal have been completed (Fig. 2).

With a special sharp-pointed Morse* scissors (Fig. 3), which also has slightly sharpened external edges, a longitudinal stab wound is made into the now exposed septal portion of the left atrial wall (Fig. 4). The blades are scissors are spread to make a longitudinal slit which will allow entrance of the tip of the left index finger.

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*Obtainable from Geo P. Pilling & Son Co., Philadelphia, Pa.

The operator's ungloved left index finger is bathed in a dilute (1/10,000) heparin solution and is inserted, palmar surface upward, into the incision in the atrial wall tamponading the bleeding which also is restrained by slight tension applied to the purse-string suture. The finger tip is advanced gently, the tourniquet tension being modified appropriately.

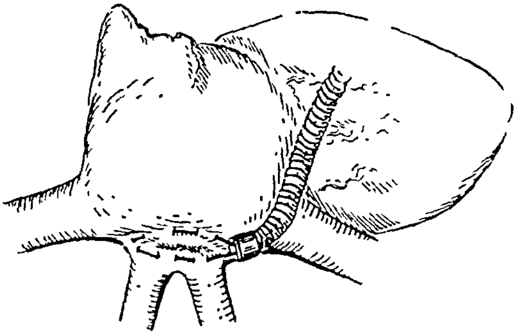


Fig. 2.—Lateral view of right side of heart with the patient face up, showing the location of the purse-string suture about the interatrial groove.

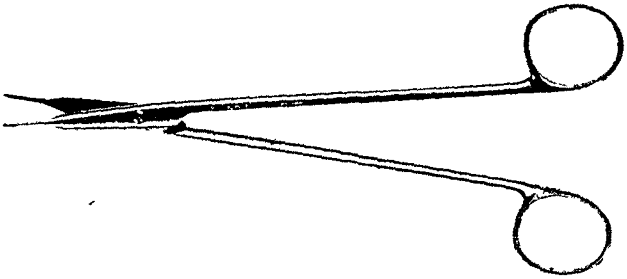


Fig. 3.—Photograph of Morse scissors.

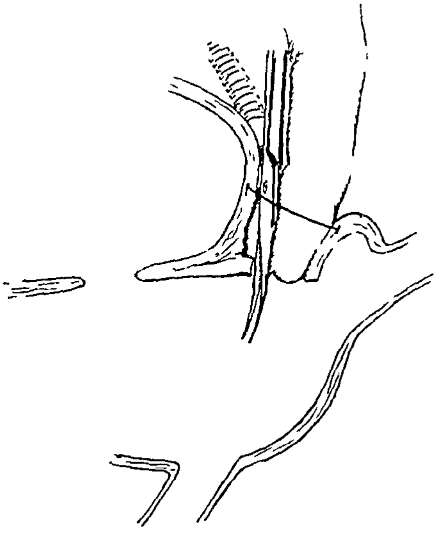


Fig. 4.—Transverse section showing entrance of pointed scissors followed by finger into left atrium in the posterior portion of interatrial groove.

After careful palpation of the septal defect, all normal or abnormal pulmonary vein ostia, the mitral and tricuspid valves, and the vena caval and coronary sinus ostia, the right atrial wall is compressed down upon the defect by the operator's left thumb (Figs 5 and 6). The intracardiac left index fingertip holds the septal margin up against the partially invaginated left atrial wall. Provision is made for preservation of an adequate intra-atrial passageway for both the superior and inferior vena caval blood flow to the tricuspid valve This passage may be placed either anterior or posterior to the region

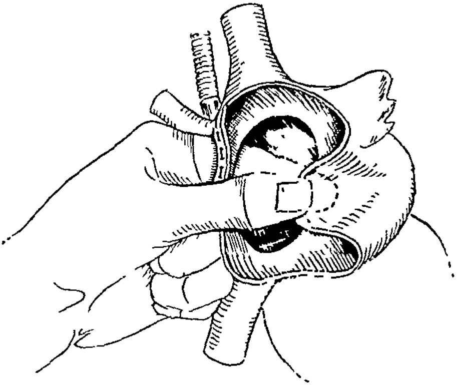


Fig 5 —Anteroposterior view showing approximation of right atrial wall to the edge of the defect by the intracardiac (left atrial) index finger apposing the extracardiac thumb

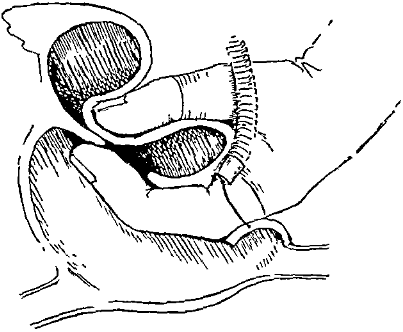


Fig 6 —Cross section showing approximation of right atrial wall to the edges of defect

of atrioseptal approximation, whichever is more convenient In some cases, when the defect lies centrally, both an anterior and a posterior channel for blood flow is provided (see Fig 1, *A*).

A heavy nylon suture on a small curved Atraumatic needle is passed through the external atrial wall into the atrial lumen, then, through the adjacent margin of the septal defect and out again through the atrial wall, forming either a simple or, preferably, a mattress type of approximating stitch. This suture is easily placed by the guidance of the intra-atrial index finger. The actual or near approximation of the structures being sutured renders facile the passage of the needle (usually in a single arcuate movement). Preferably, each successive suture is applied into the margin of the defect in an overlapping fashion, so that one half of that portion of the septal and atrial walls approximated by the previous suture is also included in the subsequent one. Several such sutures may be placed before any are tied. When they are drawn down together, the distribution of tension tends to prevent any tearing out of the sometimes delicate septal edge.

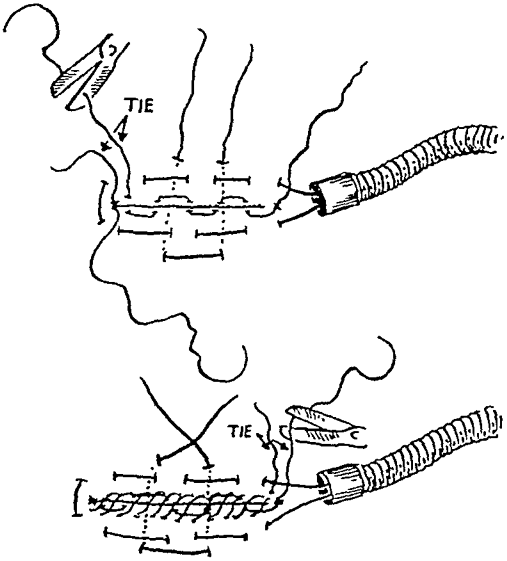


Fig. 7.—Closure of dissected interatrial groove with continuous everting cotton sutures.

After the septal closure has been accomplished, as the intracardiac finger is withdrawn, the original nylon everting mattress suture placed across the dissected interatrial grove is tightened. This eversion ensures the endocardium-to-endocardium approximation of the lips of the left atrial incision which is necessary to prevent the formation of an intra-atrial thrombus. The two external lips of the dissected interatrial groove are secured with a row of continuous everting cotton sutures and a second row of running over-and-over sutures, employing the previously placed stay sutures which have been left long with the needles attached (Fig. 7).

TABLE II. CONCOMITANT LESIONS IN 83 CASES OF ATRIAL SEPTAL DEFECT (SECUNDUM TYPE)

LESION	NUMBER OF CASES	PERCENTAGE
Uncomplicated (by other cardiac lesion)	59	71.1
Associated anomalous pulmonary venous drainage	15	18.1
Associated mitral stenosis	8	9.6
Anomalous veins and mitral stenosis	1	1.2
Total	83	100.0

MODIFICATIONS OF TECHNIQUE (Table II)

Coexisting Anomalous Right Pulmonary Venous Drainage.—The orifices of any such veins which enter directly into the right atrium may be diverted into the left atrium by invaginating the vein-bearing portion of the right atrial wall against the usually conveniently located septal defect. If the defect does not lie in close proximity to the mouths of the anomalous veins, one may be made in an appropriate location by deliberately incising the septum. Then, the vein-bearing portion of the atrial wall may be sutured against the region of the created septal defect (Fig. 8). In either event, the anomalous veins are caused to drain directly into the left atrium while the vena caval flow is separated from it. A similar technique may be used for the correction of anomalous drainage of the right pulmonary veins in the absence of any septal defect.[19]

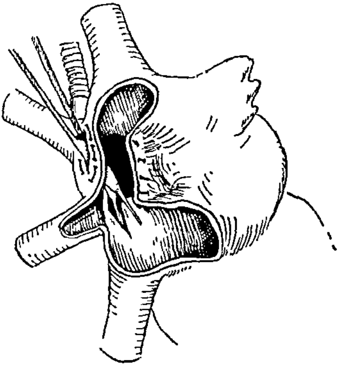


Fig. 8—The creation of a septal defect for drainage of anomalous pulmonary veins by means of special intracardiac scissors

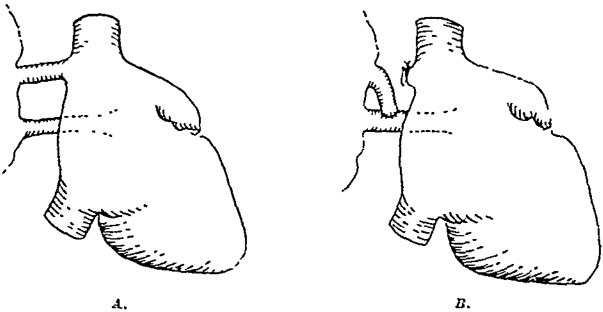


Fig. 9—*A*, One right pulmonary vein anomalous (into superior vena cava), the other normal

B, Surgical correction by implantation of end of divided abnormal vein into side of normal vein

Should one or more of the right pulmonary veins enter directly into one of the venae cavae, one of the several logical modifications of these techniques will present itself. If one of the right pulmonary veins enters the left atrium, the other (anomalous) one may be disinserted from the vena cava and anastomosed end-to-side to the normally draining vein (Fig. 9). Should one be anomalous into the right atrium while the other drains into the vena cava, the latter may be detached and anastomosed to the former. Then, atrioseptopexy may be carried out with application of the vein-bearing portion of the right atrial wall against the pre-existing or created septal defect.

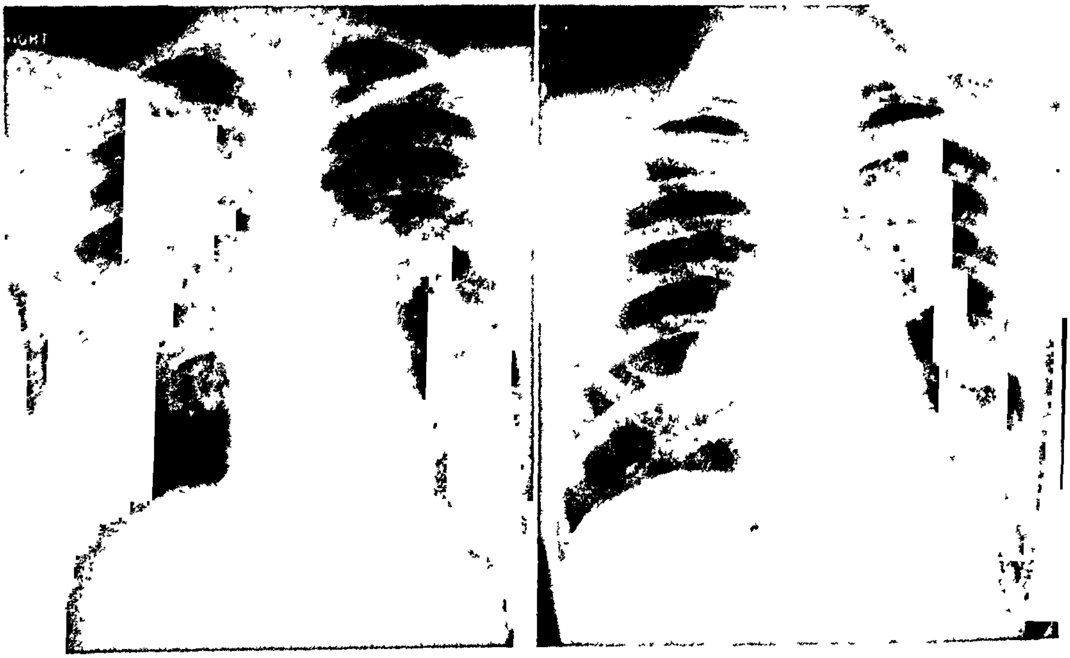
In case of total anomalous pulmonary venous drainage, the then necessarily present defect and the anomalous right pulmonary veins should be corrected at once. Retransposition of the anomalous left pulmonary veins may be performed at a later operation.

We feel that normal lung tissue should not be resected simply because of the existence of anomalous venous drainage unless there is no other feasible solution.

Coexistent Mitral Stenosis.—We now feel that a stenotic mitral valve may be more effectively treated by a right-sided approach[17, 18, 20, 21] than by the usual left-sided one, because the technically easier access to *both* commissures facilitates a more complete valve mobilization. From the right side, one is directly above the valve and in line with the left atrioventricular channel, whereas, when the mitral valve is attacked from the left side via the left auricular appendage, it can only be done with a finger bent back on itself or by the use of a valvulotome with an appropriate extreme curve or angulation.

ILLUSTRATIVE CASE REPORT

G. B. (No. 56-264439)), a 20-year-old white man, had been known to have a heart murmur since the age of 13. He had been almost free of symptoms up to the time of



A. *B.*

Fig. 10.—X-rays of G. B. *A*, Preoperative chest x-ray. *B*, Postoperative chest x-ray.

admission, except for some fatigability and some shortness of breath on heavy exertion There was a harsh Grade 3 systolic murmur at the left third intercostal space near the sternal border The pulmonary second sound was accentuated The blood pressure was 120/80 mm Hg There was no cyanosis and no clubbing of the fingers or toes

By x ray, the heart was moderately enlarged (Fig 10) with a prominent pulmonary artery and increased vascularity of the lungs The electrocardiogram showed a normal sinus rhythm at a rate of 80, with right ventricular hypertrophy and strain Cardiac catheterization revealed a left to right shunt at the atrial level of 6 8 L per minute, with a normal pulmonary artery pressure

Surgery was performed on Oct 17, 1956 The right atrium was hugely dilated The septal defect was a very large central one of the secundum type There was a small (5 to 8 mm) ridge of tissue about the periphery The defect was approximately 5 to 6 cm in diameter and oval Its lower (caudal) margin lay about 5 cm medial to the lateral atrial wall It was successfully and easily closed by the above described technique, the intra cardiac index finger lifting the septal margin as the thumb depressed the lateral atrial wall to make contact with it The time which elapsed from the insertion of the finger to closure of the defect was 22 minutes The postoperative course was completely uneventful At the present time, no murmurs are audible

Since that time, 16 additional patients with ostium secundum defects have been treated easily and simply by this modified technique without operative mortality

DISCUSSION

At the present time, three general plans of attack are available for closure of atrial septal defects They are—the open, the semi open ("well"), and the closed techniques The open techniques entail the use of a pump oxygenator, cross circulation, or hypothermia It is our opinion that these open surgical techniques are associated with greatly added risk and that their use is not justified for the closure of simple ostium secundum defects The semi open technique of Gross as elaborated by Kirklin seems to us to be unnecessarily complicated and time consuming for closure of a simple defect Gross's recent adoption of the technique of atrioseptopexy would seem to imply a similar conclusion

Ostium primum defects would seem to require an open technique for their closure Defects with reversal of shunt represent an end stage of disease which may preclude correction by surgery Associated lesions, such as mitral stenosis or anomalous drainage of the right pulmonary veins, may be treated definitively at the time of correction of a secundum type defect when the closed method of atrioseptopexy is employed

SUMMARY

Simplicity, wide applicability, and safety to the patient distinguish the technique of atrioseptopexy as originally described An open technique seems indicated for the correction of the less common and more difficult ostium primum type of lesion

A modification of the technique of atrioseptopexy is suggested in which the intracardiac finger is introduced by way of a right sided thoracic incision directly into the *left* atrium, so that it lies behind the interatrial septum, thereby considerably facilitating the steps of closure of the defect

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VENTRICULAR FIBRILLATION IN HYPOTHERMIA

A REVIEW OF FACTORS FAVORING FIBRILLATION IN HYPOTHERMIA WITH AND WITHOUT CARDIAC SURGERY

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IT IS generally agreed that ventricular fibrillation (VF) constitutes the most serious complication of profound hypothermia. It is still the main deterrent to the more extensive use of hypothermia for various surgical interventions despite the claim that it may be used to advantage in cardiac surgery.[1-4] In order to prevent this arrhythmia during hypothermia, particularly in cardiac surgery, it is necessary to know the factor(s) which induce it under such circumstances. Unfortunately, in spite of much work, there is still no general agreement about the exact mechanism of the onset of VF under normothermic conditions. The simplest working concept may be briefly outlined as follows. An ectopic focus or foci in the ventricles start discharging repetitively at a high and accelerating frequency (the high frequency is relative rather than absolute, being related to the refractory period of the muscle). If one of these impulses is discharged very soon after another it may be blocked on one side, provided it falls in the relative refractory period of the preceding impulse. However, the impulse traveling in other directions may proceed over the ventricular syncytium if the muscle has just recovered its excitability or is in a hyperexcitable state. Conditions are now favorable for re-entry and spread of excitation occurs in a very irregular and haphazard fashion depending upon the availability of excitable tissue. As a consequence, the ventricular contractions are fasciculated, producing the characteristic fibrillatory movements.

During hypothermia the physiology of practically every system in the body of the nonhibernating homeotherm is profoundly altered and it is the purpose of this review to analyze the role of these changes in favoring the onset of VF.

I. *Influence of Anesthetics.*—

Covino, Charleson, and D'Amato[5] noted that cardiac arrhythmias and VF occur less frequently in profoundly hypothermic dogs under thiopental or ether than under pentobarbital, whereas, in normothermic animals, arrhythmias are more frequent under Pentothal anesthesia. Riley and associates[6] similarly reported that there is a lower incidence of VF with thiopental anesthesia compared to pentobarbital.

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II. *Role of Cardiac Nerves.*—

The influence of extrinsic cardiac nerves on the VF of hypothermia is a moot question. Shumacker and colleagues[7] showed that bilateral sympathetic denervation of the heart or the use of a sympathetic ganglionic blocking agent (Arfonad) prevented fibrillation in hypothermic animals subjected to inflow occlusion and right ventriculotomy. On such grounds, Riberi,[8] Radigan[9] and their co-workers have used procaine infiltration of the junction of the right atrium with the superior vena cava and reported protection from VF during hypothermia and right ventriculotomy. Contrary to these observations, Covino and co-workers[5] found that adrenergic blockade with Dibenamine or SY-21 offered no protection against hypothermic VF in the absence of cardiac surgery.

Shumacker[7] noted that bilateral cervical vagotomy offered no protection. On the contrary, electrical stimulation of the undivided right vagus nerve below the level of the caudate ganglion reduced the incidence of VF. A similar result was noted by Montgomery, Prevedel, and Swan[10] who utilized vagal stimulation to prevent VF during catheterization of the coronary sinus in hypothermic animals.

From these studies one is led to conclude that increased sympathetic activity and perhaps decreased vagal tone on the heart tend to favor the onset of VF. Whether or not such a change in the activity of cardiac nerves prevails during hypothermia is uncertain, although some investigators[10] postulate that it does. This is thought to be a consequence of sino-aortic reflexes which may be activated from the fall of arterial pressure that occurs in hypothermia. The situation is actually much more complex than is implied by this hypothesis because there is some evidence that hypothermia affects the discharge of baroceptors as well as that of the medullary cardiac centers. Furthermore, the action of the neurohumoral agents (epinephrine and acetylcholine) released by the postganglionic nerve endings is increased in cold. Fuhrman and colleagues[11] showed that injected epinephrine causes an increase in the amplitude and duration of contraction of the denervated nictitating membrane at lower temperatures. This is explained by the action of cold in retarding the rate of enzymatic inactivation of epinephrine. Garb and Penna[12] showed that in isolated cat auricles, epinephrine produces a proportionately greater increase in rate and amplitude at 29° C. than at 37° C. Similarly, Jourdan and Marduel[13] have noted that vagal inhibition of heart lasts much longer in dogs at 30° C. than at 37° C., probably because enzymatic hydrolysis of acetylcholine is delayed by cold (acetylcholine esterase is less active at low temperatures). Some have erroneously interpreted this result to imply increased vagal tone during hypothermia.[14]

In contrast to the above results, the work of Reissmann and Kapoor[15] suggests that the extrinsic cardiac nerves play little if any role in precipitating VF in profoundly hypothermic dogs. They noted that the terminal hypothermic temperature at which VF supervenes is about the same in the denervated heart-lung preparation as in the intact dog. This comparison may be unjustified, however, on the grounds that conditions in the two preparations are not the same and that the interplay of different factors may have produced VF at the same temperature. It would have been better to compare intact animals with others in which the hearts were previously denervated and the adrenals inactivated.

III. *Role of Blood Changes.* —

a. *Hormones:* Berne[16] has shown that the administration of epinephrine to the hypothermic heart precipitates VF. This is in line with the observation of Shumacker[7] that cardiac sympathectomy partially protects the heart from VF. Brown and Cotten[17] have noted that in hypothermic dogs there is an increase in the levels of circulating epinephrine and norepinephrine. Whether or not such increases play a role in precipitating VF in profound hypothermia remains problematic. From Reissmann's[15] studies, it appears that such factors are not involved. However, when cardiac inflow and outflow vessels are occluded in hypothermic animals or man, there may well be an appreciable accumulation of catechol amines in the inferior caval blood as a consequence of sino-aortic reflexes, and this may partly explain the high incidence of VF soon after the release of occlusion. On the basis of such a hypothesis, Cookson, Neptune, and Bailey[18] used a quick acting adrenergic blocking agent (Benodaine) which markedly reduced the incidence of VF following the release of venous occlusion. In contrast, Covino[5] observed that adrenergic blockade with Dibenamine or SY-21, given immediately before immersion, failed to afford protection against VF in severely hypothermic animals.

Recently, Watkins and colleagues[19] have shown that premedication with somatotrophin reduces the incidence of VF during cardiac surgery in hypothermic dogs as well as the VF produced by the ligation of a coronary artery in normothermic animals. The mechanism of action remains unknown. In this connection it would be of interest to know whether or not hypothermia alters the amount of circulating somatotrophin.

b. *pH changes:* It has been shown that hypothermia induces a respiratory as well as a metabolic acidosis in anesthetized animals breathing spontaneously. Brown and Miller[20] noted that normothermic dogs, made hypercapnic by breathing 30 to 40 per cent CO_2 for long periods, developed VF if suddenly placed on room air and hyperventilated. Swan and colleagues[21] presented data which suggested that VF in hypothermic dogs may be initiated by a sudden rise in blood pH from abnormally low levels. This was avoided to a certain extent by vigorous hyperventilation with oxygen to a state of alkalosis during the period of cooling and has been widely used by cardiac surgeons. Studies in the heart-lung preparation suggest that alkalemia does not play a role in precipitating or preventing VF in profound hypothermia.[22]

On the other hand, Moulder and co-workers[23] noted that a state of alkalosis is associated with considerable VF before surgery is done in hypothermic animals. These investigators recommend self-ventilation during cooling and minimal respiratory assistance after thoracotomy so as to maintain the blood pH near normal. Other workers[24] claim that ventilating the lungs with 2.5 to 11.5 per cent CO_2 in oxygen helps to prevent VF. Thus, it is clear that the problem is by no means settled.

Covino and Hegnauer[25] had emphasized the importance of acidosis in hypothermic VF and, in their preliminary studies, had suggested that acidosis favors VF by increasing the excitability of ventricular myocardium. However, in a more recent work the same investigators[26] showed that the earlier results were due to a technical error and that actually there were no changes in ventricular thresholds during hypothermia. Montgomery, Prevedel, and Swan[10] suggested that acidosis might possibly act by changing the potassium concentration in myocardial tissue. They noted that the hypothermic heart during respiratory acidosis gains K, whereas during respiratory alkalosis it maintains K balance.

c. *Plasma electrolytes:* Much interest is centered on the possible role of alterations in the concentration of plasma electrolytes on the onset of VF or cardiac asystole in hypothermia. Most investigators find no significant change in plasma sodium, chloride, phosphorus, and magnesium during hypothermia.

The cations of greatest interest are calcium and potassium because of their well-known effects on cardiac activity. In hypothermic dogs, Fleming[27] found no significant change in serum calcium whereas Smith[28] reported a 22 per cent increase. Elliott and Crismon[29] noted a slight increase in serum calcium in hypothermic rats that were shivering.

The studies on serum K are more inconsistent. Swan[21] and Osborn[30] found a decrease in K during hypothermia in dogs. Fleming[27] and Deterling and coworkers[31] reported no significant change whereas Smith[28] noted an increase of 45 per cent. Elliott and Crismon[29] found a marked rise of K in hypothermic rats undergoing shivering. This hyperpotassemia was attributed to hepatic glycogenolysis which is accompanied by the release of K. Whether the discrepant results in anesthetized dogs can be due to different degrees of shivering is difficult to know but appears unlikely.

In most of these studies, no correlation was made with terminal cardiac events. Hence, it is impossible to state whether or not these electrolyte changes are causally related to the onset of VF. Some investigators[32] have speculated that in hypothermia a rise of serum calcium/potassium ratio may be responsible for the onset of VF. However, the recent observation of Smith,[28] that the increase in potassium is proportionately greater than that of calcium, speaks against such a view. It may be significant to note that the hypothermic heart is more sensitive to K than the normothermic.[29]

d. *Shift of oxyhemoglobin dissociation curve:* Cooling the blood displaces the hemoglobin dissociation curve to the left, thus hindering the release of oxygen in systemic capillaries. On this basis, some workers[33, 34] postulated that hypothermia might induce hypoxia of all the tissues in the body including the myocardium. It was believed that both hypothermic VF and cardiac asystole were due to such myocardial hypoxia. However, Penrod[35] and Hegnauer and associates[36] showed that in the presence of adequate arterial saturation, coronary A-V oxygen difference remains unchanged at temperatures as low as 20° C. or 17° C. Studies by Badeer[22] demonstrated that in the heart-lung preparation increasing the amount of oxygen physically dissolved in the blood does not lower the hypothermic temperature that induces VF when cardiac manipulation is avoided. Also, Berne[16] has shown that increasing the coronary flow, up to ninefold, in a hypothermic heart does not improve the performance of the heart as judged by pressure contours. These studies demonstrate beyond doubt that the leftward shift of the dissociation curve by cold does not induce hypoxia of the myocardium provided arterial saturation is normal.

e. *Arterial hypoxemia:* Beck and Leighninger[37] reported that in normothermic animals a progressive decrease in arterial saturation causes cardiac arrest by asystole and not by VF. It was explained that such a heart is "electrically stable" because hypoxemia affects the entire ventricular myocardium equally. Similarly, in fatal hemorrhage the entire heart is hypoxic and stops in asystole.

On such grounds one would expect that should hypoxemia prevail during hypothermia it would not contribute to the onset of VF.

IV. *Influence of Work Load of Heart.—*

Bigelow and co-workers[38] reported that VF in hypothermia was associated with a high central venous pressure and that it could be prevented by venesection. Covino and colleagues,[5] on the other hand, found that venesection did not prevent VF and concluded that cardiac overloading is not a contributing factor. This is in line with the finding of Reissmann[15] who noted no relationship between cardiac load and susceptibility to VF in the heart-lung preparation, except when the load was extreme and caused a relative deficiency of oxygen supply. Such a discrepancy between load and oxygen supply may arise on the sudden release of cardiac inflow occlusion under hypothermia because the heart is already hypoxic due to the occlusion. Therefore, release must be done in stages to avoid overloading.

V. *Direct Effect of Lowered Temperature on Heart Muscle.—*

a. *Excitability:* As already pointed out, the recent work of Hegnauer and Covino[26] shows that there is no change in the intensity of threshold stimuli applied to the hypothermic ventricular myocardium at different periods of the cardiac cycle even in the presence of acidosis. Hence, it is misleading to speak of an increased "irritability" of the ventricles during hypothermia.

Studies by Coraboeuf and Weidmann[39] on single Purkinje fibers, utilizing the intracellular recording technique, showed that the magnitude of resting potential and of the spike potential is unchanged between 40° C. and 25° C., but the duration of all the phases is prolonged with fall of temperature. This result may be interpreted to indicate a slowing of the speed of migration of ions during reversed polarization (depolarization) and repolarization of Purkinje tissue, but the magnitude of ionic migration (number of ions) seems to be unchanged. Similar results were obtained on single fibers of frog ventricle and cat auricle. These findings at the cellular level agree with the latest report of Hegnauer[26] that there are no changes in the threshold of excitability of dog ventricle during hypothermia, assuming that polarization phenomena represent excitability.

We may conclude, therefore, that changes in myocardial excitability are not involved in hypothermic VF.

b. *Speed of conduction, contraction, and relaxation:* All these events are slowed during hypothermia. Slowing of conduction is indicated by the well-known prolongation of the P-R interval, widening of QRS, elevation of S-T segment and changes in the contour of P and T waves.[10]

Ventricular contraction and relaxation phases are markedly prolonged during hypothermia.[41] These changes are accompanied by lengthening of both the absolute and relative refractory periods of the ventricle.[42] Such findings agree with the observation in single fibers that all phases of electrical activity are prolonged with fall of temperature.[39]

c. *Temperature gradients:* Hegnauer[43] pointed out that during hypothermia and rewarming one would expect significant temperature variations in the ventricular myocardium and that such gradients may be responsible for ectopic activity in the ventricles. Pinkston and associates[42] have recently demonstrated the existence of such gradients by thermocouples placed at various parts of the heart. These gradients could readily produce differences in the relative refractory period in different parts of the ventricular myocardium and thus play an important role in precipitating VF by blocking an ectopic impulse in some areas and allowing it to travel in others.

d. *Ventricular ectopic activity:* Experimental evidence indicates that at temperatures below 25° C. there is a tendency for the development of ectopic foci in the ventricles. Since the pathophysiology of ectopic discharges is still not well understood it is difficult to state the mechanism of ventricular ectopic activity in hypothermia. According to Di Palma,[14] acute failure of sinus activity may be a cause of ventricular rhythms. These ectopic discharges, specially when accelerating, may be blocked unilaterally as a consequence of differences in the refractory period and thus initiate re-entry and VF. It is likely that such discharges come during the so-called ''vulnerable period'' of ventricular activity (end of systole) in order to cause fibrillation. Many workers suspect that changes in pH and/or K of ventricular myocardium are somehow involved in ectopic activity and the onset of VF.

e. *Myocardial electrolytes:* Montgomery and co-workers[10] reported that in hypothermic acidotic animals which terminate by VF, the myocardium gains K prior to the arrhythmia, whereas in alkalotic animals there is no change. Later studies by Covino and Hegnauer[44] gave different results: In hypothermic acidotic dogs which terminated by VF, there was a loss of K and H ions from cardiac muscle and a gain of calcium. However, in those animals that did not end by VF, the myocardium neither gained nor lost these ions. The authors explain the discrepant results of Montgomery and co-workers[10] on the basis that these workers used vagal stimulation or acetylcholine or Prostigmin in their experiments and that these chemicals are known to alter the K balance of the heart. Recently, Smith,[28] in agreement with Covino and Hegnauer,[44] also noted a loss of K from heart muscle just prior to VF in many of his experiments. Currently much interest is centered on the role of K but more research is needed to assign its exact role.

Covino and Hegnauer[44] reported that there was no gain or loss of myocardial sodium, chloride, and magnesium in hypothermic fibrillators or nonfibrillators.

VI. *Mechanical Stimuli.—*

It is well known that manipulating, clamping, or cutting the heart, as well as the presence of intraventricular catheters act as mechanical stimuli even under normothermic conditions. In normothermia such stimuli rarely induce VF. In the cold state, on the contrary, such stimuli are potent factors precipitating VF.[15, 36] The difference probably lies in the slowed conduction and variations in the refractory period of different parts of the hypothermic myocardium.

VII. *Coronary Blood Flow.—*

All investigators agree that in hypothermia there is a reduction in coronary flow.[16,45-47] Although Smith[28] claims that the decline in coronary flow leads to hypoxia of the heart and is a factor in the onset of VF, the majority of workers[16, 22, 35, 36, 47, 48] agree that there is no evidence of myocardial hypoxia in hypothermia. The oxygen demand of the heart is greatly reduced by the low myocardial temperature, the reduced cardiac output and arterial pressure and the bradycardia. All these cut down the demand for oxygen so much that the curtailed coronary flow presumably remains adequate for the requirements of the myocardium. Hence, VF during profound hypothermia, in the absence of cardiac manipulation and inflow occlusion, is not due to myocardial hypoxia.

In cardiac surgery with inflow occlusion under moderate hypothermia, the situation is somewhat different. The ventricles are now subjected to a diffuse ischemia and the question arises whether or not such ischemic hypoxia contributes to the onset of VF which usually develops some time after occlusion. If Beck's view (see *Arterial Hypoxemia*) is correct, such diffuse ischemia should not play a role in VF. However, Edwards,[49] Lewis,[50] and their associates showed recently that perfusion of the coronaries with oxygenated blood during the period of occlusion markedly reduces the incidence of VF. These reports suggest, contrary to Beck's hypothesis, that diffuse hypoxia of the myocardium may be a factor leading to VF, at least in the hypothermic heart.

CONCLUSION

Ventricular fibrillation in hypothermia with and without cardiac surgery is not due to the operation of any one factor. The tendency for the development of accelerating discharges from ectopic centers in the ventricles coupled with prolongation and variation in the relative refractory period produced by the lowered temperature seem to be the most important factors initiating VF. Conditions that favor ventricular ectopic discharges, such as, changes in blood pH, disturbed myocardial Ca-K balance, mechanical stimuli, increased activity of cardiac sympathetics, increased amounts of circulating catechol amines, interference with coronary arterial flow, anesthetics and drugs, etc., favor the onset of VF during hypothermia. It appears that prolongation of the refractory period due to cold per se and differences in the refractory period due to temperature gradients pave the way for the onset of VF.

Prevention by physiologic and pharmacologic means should aim at reducing or eliminating these factors as much as possible.

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CARCINOMA OF THE LUNG TREATED BY LOBECTOMY: THE PROBLEM OF LOCAL RECURRENCE

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AS THE pendulum swings from the more radical extirpations of carcinoma of the lung to lesser resections, the problem of local recurrence assumes a more prominent role. This recurrence, be it in the bronchial stump or in the hilar lymph nodes, is a poor reflection on the operative procedure, especially if it was an elective lobectomy or bilobectomy. Pneumonectomy, bilobectomy, and lobectomy in selected cases of carcinoma of the lung have yielded fairly good results[1-3] and, at present, represents our only effective weapon against this disease. Therefore, it is felt that the underlying pathology and the pulmonary functional status of the patient should determine the extent of resection rather than enthusiasm for a particular operative procedure or the unsuppressed desire to preserve all normal lung tissue.

Recently, Habein, McDonald, and Clagett[4] reviewed 18 cases of recurrent carcinomas of the bronchial stump in a series of six hundred and thirty-one extirpations and resections for carcinoma of the lung (an incidence of 2.9 per cent). In this group there were no known recurrences in the lobectomy group and only two in the bilobectomy group. In our series of twenty-one lobectomies and bilobectomies for carcinoma of the lung, performed prior to February, 1955, there are two known recurrences (an incidence of 9.5 per cent) and 2 suspected recurrences in patients who died at home. Because of the difficulty in following all patients personally, who have had resections for carcinoma of the lung, we have considered the presence of persistent cough, hemoptysis, and inanition as evidence of recurrence until proved otherwise.

The two proved recurrences in our series of twenty-one resections (sixteen of which were for squamous-cell carcinomas) and an additional case, a patient whose lung had been resected in another hospital and who had been followed on this service until death, were all squamous-cell carcinomas. Also, the 18 cases of Habein, McDonald, and Clagett,[4] previously referred to, were all squamous-cell carcinomas.

CASE REPORTS

CASE 1. The patient, a 58-year-old white man, was admitted to the VA Hospital, McKinney, Texas, in December of 1954, for the sixth time during the past eight years for a chronic inflammation of the upper eyelid and an active duodenal ulcer. Routine x-ray on this admission disclosed an infiltrate about 5 cm. in diameter in the right lower lobe

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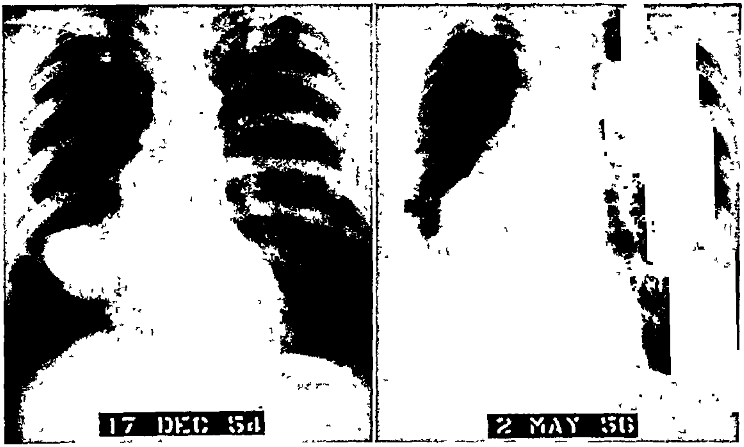
(Fig. 1, *A*) which was not present on a film taken in May of 1952. In reviewing his history, it was found that he was a heavy cigarette smoker, and had had a chronic cough for the past twenty years. During the past six months his cough had become productive and hemoptysis had occurred on several occasions.

Physical examination revealed an expiratory wheeze in the right lung field, anteriorly. He had deformities of the right leg and foot which were residuals of an anterior poliomyelitis that he had had as a child.

Clinical laboratory findings were all within normal limits. Pulmonary function studies including a total vital capacity and maximum breathing capacity were 66 per cent of the predicted normals.

Bronchoscopy disclosed a granular tumor in the basilar segmental bronchi of the right lower lobe that was reported by the pathologist to be a squamous cell carcinoma. In January, 1955, a right middle and lower lobectomy were performed. Epithelial metaplasia was found on the cut end of the bronchus; however, because of emphysema on the left side and the patient's shortness of breath, it was decided not to remove the remaining lobe on the right.



A. *B*.

Fig. 1.—Case 1. *A*, Chest roentgenogram showing the carcinoma in the right lower lobe. *B*, Postoperative recurrence sixteen months after a right middle and lower lobectomy.

He remained well other than shortness of breath and ulcer symptoms until May of 1956, at which time he was readmitted with hemoptysis of three days' duration. Chest x-ray at this time revealed a large recurrent tumor in the right lower lung field (Fig. 1, *B*). Bronchoscopy also showed tumor extending up to the carina on the right. X ray treatment was started and he was finally discharged in July, 1956, after receiving a tumor dose of 2,200 r. At present he is living a quiescent life in a government domicile.

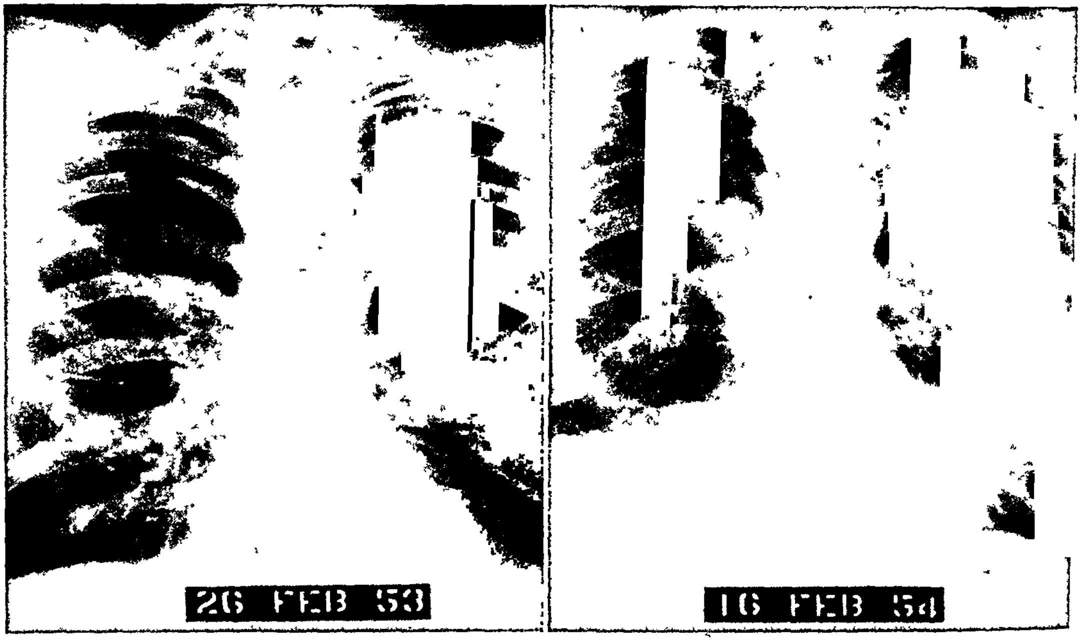
CASE 2. The patient was a 41-year-old white man admitted to VA Hospital, McKinney, Texas, in February, 1953, with a history of progressive shortness of breath since August, 1952. He smoked cigarettes heavily and had had a chronic cough for the previous twenty-five years.

Physical examination disclosed only distant breath sounds over the left apex anteriorly and expiratory râles over the entire right hemithorax.

Laboratory studies were all within normal limits. Roentgenograms of the chest revealed several thin walled cysts in the left upper lung field and a nodular lesion in the right hilum (Fig. 2, *A*). Pulmonary functions preoperatively showed the following: maximum breathing capacity, 111 L. per minute, and a total vital capacity of 1.1 L. in ½ second, 2.0 L. in one second, and 2.8 L. in 1½ seconds. There was little change after the first operation.

In March, 1953, two large blebs were excised from the apical segment of the left upper lobe and a smaller one from the lingula of the left lower lobe, following which poudrage was carried out over the left upper lobe. The patient tolerated this procedure well and in May, 1953, a right upper lobectomy was performed for a deep-seated squamous-cell carcinoma. Pneumonectomy was not performed because of the presence of bilateral emphysema and because it was felt that lobectomy had removed the tumor completely.

Pathologic sections of the cut end of the bronchus and adjacent lymph nodes revealed no evidence of tumor.



A. *B.*

Fig. 2.—Case 2 *A*, Chest roentgenogram demonstrating the carcinoma in the right upper lobe *B*, Postoperative recurrence one year after a right upper lobectomy

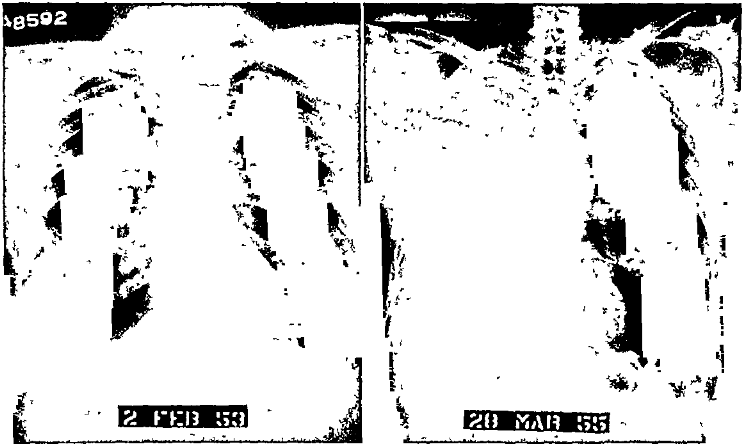
The patient did well until February, 1954, when a follow-up chest roentgenogram showed a recurrent tumor in the right hilum (Fig. 2, *B*). Bronchial secretions were positive for tumor cells. X-ray therapy was given at this time, but despite this the mass grew larger. Several other nodules developed in the right lower lobe, and the patient died with cerebral metastasis on Sept. 30, 1955. Permission to perform an autopsy was refused.

CASE 3. The patient was a 58-year-old white male salesman admitted to VA Hospital, Dallas, in October, 1954, with the complaint of a firm, tender mass in the right supraclavicular fossa. This mass had been present for three months prior to admission and was growing larger. In February of 1953, this patient had had a routine roentgenogram of the chest taken at the VA Regional Office (Fig. 3, *A*) which showed an infiltrate in the right lower lobe. He returned to a private physician who also studied him and he was told that he had a tumor in the right lung. On March 2, 1953, a right middle and lower lobectomy was performed in another hospital and he had an uneventful convalescence from this procedure until the right supraclavicular tumor manifested itself. His past history and systemic review were noncontributory.

Physical examination, on admission, disclosed several scars over his face where skin cancers had been removed, and he wore an artificial limb on the left lower leg which had been amputated, in 1922, for a chronic osteomyelitis. A firm mass about 4 cm. in diameter was present in the right supraclavicular fossa which was attached to the deep structures below it. He also had a well healed posterior lateral thoracotomy scar on the right and some limited expansion of the right hemithorax. Breath sounds on the right were somewhat limited in comparison to those on the left.

Laboratory studies revealed a slight anemia. Roentgenograms of the chest revealed an increased density in the anterior segment of the left upper lobe with several other small infiltrates below it and the usual postoperative changes in the right lung.

Biopsy of the supraclavicular mass revealed a metastatic squamous cell carcinoma which resembled the primary tumor removed from the lung. Both were poorly differentiated. X-ray therapy was administered to the right supraclavicular tumor which responded fairly well. He was discharged but was readmitted for the last time with some condylomata acuminata and another squamous cell carcinoma of the right wrist. Both of these tumors were removed without incident; however, during his hospitalization he developed a fever, and a severe cough, followed by a rapid downhill course. Chest studies disclosed a pneumonia in the right lung field with a questionable effusion. This later developed into a complete atelectasis of the remaining right upper lobe (Fig. 3, *B*). On April 4, 1955, he had two clonic convulsions and died shortly thereafter. Autopsy revealed a recurrent carcinoma of the bronchial stump on the right which had occluded the right upper lobe bronchus. There were also metastatic carcinoma sites in the left lung, right adrenal, and mediastinal lymph nodes.



A. *B.*

Fig 3.—Case 3 *A*, Chest x-ray showing the carcinoma in the right lower lobe. *B*, Postoperative recurrence with atelectasis of the right upper lobe twenty-five months after a right middle and lower lobectomy

DISCUSSION

The grave potential of a squamous-cell carcinoma of the lung is clearly illustrated in these 3 cases, and it is now our feeling that most squamous-cell carcinomas should be treated by pneumonectomy. Lobectomy and bilobectomy should be reserved for some peripherally placed adenocarcinomas, large-cell undifferentiated carcinomas, and bronchiolar carcinomas, if the hilar lymph nodes are negative for carcinoma on frozen section.

The so-called "safe margin" of 1.5 cm. of tumor-free bronchus proximal to a squamous-cell carcinoma, as described by Griess, McDonald, and Clagett,[5] has not been reliable in our experience or in that of others.[4] Frozen section of the cut end of the bronchus seems to be the most dependable method of ascertaining the presence of tumor cells or metaplasia. Epithelial metaplasia has been found to be an ominous sign and probably all suspected cases of carcinoma of the lung should have multiple biopsies of the lobar bronchus and paracarinal area, as suggested by Rabin, Selikoff and Kramer,[6] to determine their operability.

SUMMARY AND CONCLUSIONS

1. Two proved cases and two probable cases of recurrent squamous-cell carcinoma of the bronchial stump occurred in a series of 21 lobectomies for carcinoma of the lung.

2. The exact incidence of this condition is probably underestimated because of the difficulty in following all patients in person. The symptoms of a persistent cough, hemoptysis, and inanition are considered evidence of bronchial stump recurrence until proved otherwise.

3. The prognosis of a local recurrence is poor. Radiation therapy has been of little or no help and further surgery was considered meddlesome.

4. It is felt that the underlying pathology and pulmonary functional status of the patient should determine the extent of resection rather than enthusiasm for a particular operative procedure or the unsuppressed desire to preserve all normal lung tissue.

5. For the most part, squamous-cell carcinomas should be treated by pneumonectomy.

6. Lobectomy and bilobectomy should be reserved for some peripherally placed adenocarcinomas and large-cell undifferentiated carcinomas if the hilar lymph nodes are negative for carcinoma on frozen section.

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WOUND SURGERY ENCOUNTERS A DILEMMA

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> The object of this Society shall be to encourage investigations of the physiology and pathology of the thorax and its contents, to stimulate the study of the etiology, symptomatology, pathology and treatment of diseases affecting the thorax and its contents, and to disseminate such knowledge. (Article II, Section 1. Constitution of The New York Society for Thoracic Surgery.)

IF MICHELANGELO had fallen from the scaffolding while decorating the ceiling of the Sistine Chapel it is likely that he would have been treated by John of Vigo. John of Vigo was the first surgeon to record his views on gunshot wounds of the chest, and in fact his book *Practica Copiosa* printed in Rome in 1514 was one of the very early surgical texts to mention wounds by firearms. John of Vigo enjoyed a rich experience in wound surgery while attached to that fighting Pope, Julius the Second, and a nice legend perpetuated by Sir Clifford Allbutt places a French translation of the *Practica* in the hands of the young barber's apprentice, Ambroise Paré. At any rate, Paré, in 1575, quoted John of Vigo on the management of wounds of the chest.

At first thought it might seem that the treatment of an open pneumothorax would have been a straightforward matter to the ancient wound surgeons. Such a wound presents what might appear to be a simple choice between two procedures: the wound can be closed or it can be left open. As a matter of fact, the several factors which are determinants to this decision puzzled surgeons for centuries and still crop up in one form or another in our own century. In my title I have referred to this choice as a dilemma and propose to identify some of the circumstances which made the choice between leaving the wound open and closing it a difficult one.

Address at the Fortieth Anniversary Dinner of The New York Society for Thoracic Surgery, November 8, 1957.

Paré identified two major considerations. "John of Vigo," he wrote, "said that there is a disagreement between the surgeons because some have a mind to close the penetrating wound as soon as possible . . . for fear that the cold air would penetrate into the heart and that vital spirits would depart and vanish; the others, contrary minded, recommend keeping the wound open . . . in order to evacuate the blood left in the thorax, fearing that this would decompose and putrefy; that would provoke fever, fistula and other complications. In truth, those who are of the mind to close the wound immediately . . . are entirely right, on condition that there is no blood or only a small amount inside the chest. . . . In the same manner are those quite right who have the mind to keep the wound open . . ."

It is significant that Paré set out to define certain conditions or principles which were to distinguish between the two courses of action and point to the selection of the one better adapted to the particular circumstances. His text was written for the benefit of barber surgeons who, as workers in a practical art, desired to be guided by definite rules and precepts, preferably phrased in the imperative. They expected to be told either "leave the wound open" or "close the wound." Such workers are particularly unhappy when the exercise of choice between alternative methods of procedure rests on conditions that they themselves may be unable to recognize. The condition which determined that the wound should be left open was indeed definite even though difficult to assess—blood in the chest. The concept of what would happen if blood were closed in the chest was straightforward and empirical. The blood would "decompose and putrefy" and would "provoke fever, fistula and other complications."

Paré was on less certain ground when he attempted to define the conditions under which the wound should be closed immediately. He may have known that he was on unfamiliar ground, for he repeated the caution that there must be no blood or at least only a small amount of blood inside the chest if this course were to be elected. Then departing from his own surgical experience, Paré invoked a prevailing doctrine of ancient authority. "Close the wound," he wrote, "for fear that the cold air will penetrate into the heart and the vital spirits depart and vanish."

So the dilemma presented by an open chest wound as seen by the great surgeon of the Renaissance was: surgeons who close the wound to keep the vital spirits from departing may be right but experience shows that if blood remains in the chest, mischief will follow. Judging from the case reports of his own practice, it was Paré's custom to keep the wound open 2 or 3 days and allow the blood to trickle out by gravity; when the flow of blood ceased, the wound was closed. This compromise undoubtedly reflects Paré's estimate of the relative validity of the indications for the choice between closing the wound and leaving it open under the circumstances which actually prevailed. It is likely that the wounded of that period who survived to come under treatment by a surgeon had wounds of small cross-sectional area resulting from sword thrusts or low velocity missiles.

Let us now briefly trace the development of the more precise understanding of the two courses of action that confronted Paré as the two horns of a dilemma, first taking up the matter of blood in the pleural cavity. The untoward effects of the accumulation of blood in any wound are related in the legends of ancient wars and many romantic stories can be found of friends who, having put their mouths to the flesh, sucked blood from the wounds of warriors. In the wars of the seventeenth and eighteenth centuries the sucking of wounds was so much in demand that it became a trade. Certain individuals in a regiment, frequently the drummer, became famous for their skill as wound suckers. There is adequate testimony in the old writings to establish the efficacy of this form of treatment, which of course is in principle a sound method in the light of present day knowledge. Sucking was found particularly useful when quarrels were decided by duels with swords which terminated with the first flesh wound. The wounded swordsman after this form of treatment commonly was able to walk home and entirely conceal the affair. Today it is recognized that contaminated blood remaining in a wound track offers an inviting pabulum for the growth of pathogenic bacteria, and it is the usual practice with flesh wounds to leave them open with textile drainage and to close them on the fourth day by reparative suture.

The activities of wound suckers finally fell into disrepute and the procedure itself became known as the *secret dressing*. To inflate their prestige and possibly in order to keep their trade to themselves the suckers pretended to make it a magical ceremony. They muttered cabalistic words, made strange motions, and drew the sign of the cross. It was understandable that the priests who also followed armies became outraged by such profanities and refused extreme unction or other sacrament to those who participated in these diabolical ceremonies. The suckers on the other hand refused to ply their art on those who had commerce with the priests under the pretense that Christian rites interfered with their incantations. La Motte, a surgeon of this period, was an eye witness to many wonderful cures. He saw for example, a soldier who had been run through the breast with a single lunge which passed, as he described it, "in at the pap, and out at the shoulder." By examining the wounds and noticing the length of the antagonist's sword La Motte was well satisfied that the weapon had pierced the lungs and gone quite across the chest. The drummer of the regiment first sucked one wound, then turned his patient and sucked the opposite wound. He applied a piece of chewed paper upon each and next day the soldier was seen walking in the streets.

Wound sucking was thought to be particularly effective for wounds of the chest although some writers expressed doubt that retained blood actually could be removed from the pleural cavity. The question can be asked whether the phrase "sucking wound" as it is applied today to open chest wounds is a curious linguistic survival from this period because actually such a wound blows air out as much as it sucks air in.

In 1707, Anel[1] published in Amsterdam a small book on the art of sucking wounds without using the human mouth. A silver tube was the answer. One end could be attached to a piston syringe and the other applied to a wound. By this gadgetry the surgeon himself was able to take over the procedure of wound sucking, which then became known by the more refined term—aspiration. Improvements in the apparatus inevitably followed and one of these proposed by Birkholz,[2] in 1771, introduced a reservoir into the suction line, producing in essence a Potain aspirator. Anel doubted that suction applied to the margins of the wound could actually evacuate the pleural cavity and so designed a cannula patterned after a female catheter which could be introduced into the pleural space through the wound. From this it was a short step to trocar and catheter.

In 1772, Valentin[3] published his small volume in which he declared that all wounds of the chest were to be closed as a measure to suppress hemorrhage and that when necessary a counter opening should be made to evacuate the effused blood at an early date. Guthrie,[4] the great English military surgeon of the Napoleonic Wars and the Peninsular Campaign, closed wounds of the chest but followed them carefully with what he called "unremitting attention in the use of the stethoscope," and removed blood by trocar and cannula. "The first object," Guthrie wrote, "is to save life. After that, if time be given, the next will be to relieve the loaded cavity." Baron Larrey,[5] Napoleon's surgeon, supposed that 9 days should elapse before it was safe to evacuate blood without encountering the danger of renewed bleeding. If Guthrie, surgeon of the Duke of Wellington, could find an excuse to differ from Larrey he always did so; his recommended procedure was to wait until the patient reacted, when he concluded that bleeding had ceased. In following the situation he looked for the increase of serous effusion which usually came from the third to the fifth day. Then Guthrie either reopened the wound to evacuate blood or carried this out through another opening with a trocar and cannula. He placed great importance on relieving the lung from pressure at an early date so that it might expand and not be bound down by what he termed false membranes.

Having traced these significant steps in the management of the retained blood, let us examine briefly the other horn of the dilemma, namely the disturbance in pulmonary ventilation which attends an open pneumothorax. In the view of Paré, disaster resulted from the cold air penetrating into the heart and the vital spirits departing the body through the wound and vanishing. Here, as with the complication of blood accumulating in the wound, the record of experience is lost in antiquity. Take the famous case of The Dying Gaul in the Capitoline Museum of Rome dating from the second or first century B.C. (Fig. 1). Blood is pouring from the wounded chest of the warrior and his head sinks forward as his eyes dim with hypoxia. The breathing is labored and he supports his weight on a rigid arm to obtain maximal use of the unwounded lung.*

Little was understood about the lethal effects of an open pneumothorax until the early physiologists started a systematic analysis of the action of the organs of respiration. The phenomenon of pendulum air in open pneumothorax, a phenomenon which is customarily attributed in eponym to Brauer, was explained by Hoadley[6] as early as 1737.

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*Elsewhere I have given due credit to Dr. Evarts Graham for this clinical diagnosis.

> "In expiration, the air in the unwounded side was condensed, and part of it, instead of going out at the windpipe, forced its way into the lung of the wounded side, and dilated it, till the air within it came into an equilibrium with the external air which surrounded it; and in inspiration, when the air in the lung of the unwounded side became rarer than the external air, the lung in the wounded side was compressed, and part of the air within it was, by the pressure of the external air, forced back into the lung of the unwounded side, till the equilibrium was again restored."

On the basis of clinical observations, the brilliant young associate of the Hunters, William Hewson,[7] observed in 1767 that with an open external wound the patient may hardly be able to breathe but he breathes easily when it is covered. Hewson said the difficulty arose "owing to the air getting into the cavity of the thorax in inspiration instead of entering the lungs by the trachea."

UNIVERSITY PRINTS, BOSTON

DYING GAUL
CAPITOLINE MUSEUM ROME

Fig. 1.

While such a wound is uncovered the patient is deprived of the use of the lobes of that side either partially or entirely "according as the wound of the thorax bears a less or greater proportion to the branch of the trachea to that side."

These concepts based both on physiologic experiments and on clinical observations in the eighteenth century stand today except for certain amplifications and minor corrections.

It is difficult to be certain when wound surgeons closed a defect in the chest wall primarily to reestablish effective pulmonary ventilation rather than to control hemorrhage from the lung. The following story related in the memoirs of Baron Larrey, the surgeon of Napoleon, emphasizes the immediate and striking improvement in the condition of the patient that follows the closure of an open chest wound.

> "A soldier was brought to the hospital of the fortress of Ibrahym Bay, immediately after a wound . . . that penetrated the thorax, between the fifth and sixth true ribs, . . . it was about eight centimetres in extent: a large quantity of frothy and vermilion blood escaped from it with a hissing noise, at each inspiration. His extremities were cold, pulse scarcely perceptible, countenance discoloured, and respiration short and laborious; in short, he was every moment threatened with a fatal suffocation.
>
> "After having examined the wound, and the divided edges of the parts, I immediately approximated the two lips of the wound, and retained them by means of adhesive plasters, and a suitable bandage round the body.
>
> "In adopting this plan, *I intended only to hide from the sight of the patient and his comrades, the distressing spectacle of a hoemorrhage,** which would soon prove fatal: and I therefore thought, that the effusion of blood into the cavity of the thorax, could not increase the danger.
>
> "But the wound was scarcely closed, when he breathed more freely, and felt easier. The heat of the body soon returned, and the pulse rose; in a few hours he became quite calm, and to my great surprize, grew better. He was cured in a very few days, and without difficulty."

Larrey then referred to two similar cases at the hospital of the Imperial Guard.

> "Since Ambroise Paré, all practitioners and authors, who have written on wounds of the thorax, advise not to close the wounds which penetrate this cavity, especially, when they are followed by hoemorrhage. . . . But the ancients closed such wounds, and even used sutures to make them more exact. . . .
>
> "The surprising success that attended these three apparently mortal wounds . . . led me to believe that this practice is preferable to that which is now adopted."

Guthrie, whom I have already identified as the surgeon of the Duke of Wellington, remarked that Baron Larrey had received credit for advocating closure of chest wounds merely because he had so effectively supported the views of Valentin, and that although the precepts of Valentin may have been forgotten in France they were in existence and Larrey merely deserved the merit of having revived and confirmed them. Furthermore, Guthrie claimed that he himself had closed chest wounds long before he had seen any of Larrey's works and was not aware that he was acting out of the ordinary course of things except that he used a continuous suture instead of trusting to a compress and bandage. While Guthrie pointed to this as a trivial difference, far more weight would be put on this detail today, because until a surgeon can be certain that a wounded lung is not leaking air into the pleural space, the external wound should not be closed tightly with a suture A lethal pressure pneumothorax may develop within a few minutes Strapping the wound edges together as Larrey did permits the escape of air should pressure build up from a bronchopleural fistula

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*Italics are those of the author.

It was well known to Guthrie that closure of chest wounds relieved difficult breathing and he was familiar with the observations of Hewson to which he referred Nevertheless his writings give the impression that his advocacy of closure of the wound was based primarily on the belief that in accord with the recommendation of Valentin this was the most efficacious means of arresting hemorrhage from the lung Thus the two horns of Paré's dilemma became a point of issue between the surgeons of Napoleon and Wellington at Waterloo

There has been constant modification and duplication of detail in devices designed to suppress the in and out flow of air in an open chest wound Hermetical seals were tried in the Northern armies of the Civil War[8] Tampons were designed in World War I[9] and, on the Italian front, Morelli[10] used his occlusive rubber cuffs on tubes permitting suction drainage as well as closure of the defect

Further projection of the development of the understanding of open pneumothorax would lead directly into the thinking and experiments which eliminated the hazard of uncontrolled open pneumothorax during a chest operation This is a long and fascinating story of surgical technology and invention in which members of this Society and their preceptors have played an active part I mention specifically Willy Meyer, Nathan Green, O'Dwyer, Meltzer, Auer, and Lilienthal The devices developed to maintain ventilation of the lungs in the patient undergoing thoracotomy were many and varied Today the responsibility for ventilation has been assumed by the anesthetist, but for several decades this responsibility was added to that of the operation itself and assumed by the surgeon Rather than pursue the intricacies of this story,[11] it may be of greater interest to review some of the circumstances that attended World War I and surrounded the task of the Empyema Commission of 1918

A beginning may be made in 1914, 3 years before the United States entered the War At that time a fairly complete concept of the aerodynamics of pulmonary ventilation had been established, methods to maintain adequate ventilation during open chest operations had been devised, and techniques for applying these methods had been perfected It was well into 1916 before the treatment of gunshot wounds of the chest by open operation in the combat zone had been undertaken except in the German army Both French and British surgeons were submerged by an overwhelming load of casualties and preoccupied with the task of learning by bitter experience the surgical principles that must guide the management of flesh wounds from high velocity missiles and high explosive shell fragments

Then the Parisian surgeon, Pierre Duval,[12] spearheaded what appeared to be a break through on the surgical front.

> "Surgery of the lung has undergone complete revolution in the war. The lung is no longer an organ inaccessible to the surgeon. One can open the chest widely, expose the lung, lobe by lobe, manipulate it, resect it, suture it, and treat it as one would a coil of intestine in a laparotomy. Surgery of the lung does not require any of those pressure chambers which the genius of the Germans invented and their persuasian made us think necessary."

It is only fair to point out that Pierre Duval's claims were not widely accepted in France, where Grégoire and Courcoux maintained a far more conservative position. British surgeons were also skeptical although Moynihan and Gask were swept along on the tide of enthusiasm. H. Morriston Davies with his scholarly interest in thoracic physiology held to sound concepts, as did William Hutchinson of the Canadian army.

The rash claims of Duval and his disciples and their scorn of differential pressure had more repercussions in the United States than in the theater of war. In the general exuberance over all things French and the condemnation of all things German, the fact was forgotten that scientific concepts recognize no national boundaries. Lord Moynihan,[13] then Sir Berkeley, Colonel in the R.A.M.C., informed audiences in the United States that a surgeon might attack the lungs boldly, without heed to the danger of an open pneumothorax. Following the pattern of the triumphal tours of the victorious generals, Duval appeared before professional groups in the United States in the autumn of 1918. I quote from an Editorial[14] in the American Journal of Surgery:

> "Those who were fortunate enough to hear the addresses of the visiting foreign surgeons at the College of Physicians and Surgeons and at the New York Academy of Medicine in the early November days will not soon forget these memorable occasions. They were all inspiring addresses, especially in the setting of spontaneous patriotic fervor that marked those days. They were set forth with a remarkable lucidity and grace of diction; charged with big and novel surgical principles and bearing a message of profound import not only to war surgery, but to surgery of civil life.
>
> . . . we have selected for comment in this number their experiences in lung surgery, because this was perhaps the most startling feature in their addresses. This phase was beautifully set forth by Major Duval, the daring originator of the latest treatment of wounds of the chest and lung . . . no one could accuse Duval of chauvinism when he pointed out that certain German principles, such as the teaching of the exaggerated dangers of artificial [sic] pneumothorax . . . had retarded the progress of lung surgery appreciably . . . the lung, like other tissues of the body, need not be handled with the timidity with which it had hitherto been accustomed. . . . Essentially, therefore, the method devised by Duval consists in wide exposure of the chest cavity, producing a complete pneumothorax with prolapse of the lung; pulling out and exposing each lobe of the organ, and treating the wound in the same manner as a wound in any other part of the body, followed by complete closure of the thoracic cavity, without drainage."

And so patriotic fervor once again was to solve both horns of the old dilemma of John of Vigo and Paré.

In reflecting upon the narrative of the experience of wound surgeons in the 1914-1918 War as compared with that of surgeons in previous and subsequent wars, I have become increasingly convinced that the outstanding feature of this period was the prevalence of hemolytic streptococcus infection both in Europe and in the United States. This epidemic assumed pandemic proportions when the hemolytic streptococcus rode the coattails of the influenza virus which swept around the world. For over a decade after the end of the war the hemolytic streptococcus haunted the surgical wards of our civilian hospitals and made return visits, particularly during the winter months. Those of us studying surgery during this period were weaned on the surgery of acute tenosynovitis, mastoiditis, primary streptococcal peritonitis, erysipelas, and the catastrophic infections of supposedly aseptic operative fields with this organism. The streptococcal pneumonia and empyema which accompanied the influenza epidemic of 1918 was a new disease to a generation of doctors. They had no precedents which defined it. In the past three decades, as far as my own experience is concerned, the disease is, for all practical purposes, extinct. During the 1914-1918 War, however, it gave rise to the Empyema Commission, which in the minds of thoracic surgeons is identified with Dr. Evarts Graham.

Following the entrance of the United States into World War I, Dr. Graham* was on duty as a captain in the Medical Corps assigned to take a course in neurosurgery in Chicago when he was visited by Dr. Allen Kanavel who was on duty as a consultant in the Office of the Surgeon General of the Army. Kanavel told Graham that there was growing apprehension about difficulties ahead with empyema as the country was then in the first year of an influenza epidemic which would undoubtedly increase in severity—would he be interested in working on the problem? Graham knew little or nothing about chest surgery at that time but was interested in nitrogen loss from massive exudation and had been selected because of this interest and his chemical training. Shortly after Kanavel's visit, Graham received orders to Camp Lee, there to team up with Dunham, who was working on the bacteriology of the disease. Graham and Dunham were joined by Captain Bell, a chemist of the Sanitary Corps who died a few years after the war. These three formed the hard nucleus of what became known as the Empyema Commission. With the help of Kanavel, a questionnaire was issued to camp hospitals and it was found that the average mortality rates attributed to streptococcal empyema were running approximately 30 per cent. The surgical staffs of the camp hospitals were stubbornly antagonistic to members of the Commission, whom they regarded as "snoopers" from the Office of the Surgeon General. Opportunities for observation, however, could not be denied and disclosed that cyanotic patients, with pulmonary reserves crippled by massive and oftentimes bilateral bronchopneumonia, were being hurried to an operating room as soon as thoracentesis yielded fluid containing chains of streptococci. The operation was rib resection with open tube drainage. Death occurred quite regularly about a half an hour after the operation.

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*This account of the formation and activities of the Empyema Commission was related to me by Dr Graham

It will be recalled that Sir Berkeley Moynihan had in the autumn of 1917 delivered the address in which he repeated the claims of Duval that the chest cavity could be opened as readily as the abdomen. When Graham, during his visits to army camp hospitals, expressed apprehension about the open pneumothorax which attended rib resection for empyema he was asked who he thought he was to contradict the great Moynihan. When Graham cited an actual case in which a cyanotic and dyspneic soldier had died on the operating table that very morning and asked if the open pneumothorax might not have been a contributing factor, the reply was: "No, he died because we didn't operate soon enough."

Early evacuation of streptococcal pus was taught as a dogma of the surgery of the period; tendon sheaths were opened on the least suspicion of infection and oftentimes a spreading streptococcal cellulitis was slashed with multiple incisions long before fixation abscesses appeared. Graham was certain that the open pneumothorax was a factor in the deaths from empyema but at that time was not familiar with the literature on open pneumothorax, nor did he have sufficient observation or experiments to prove his point.

In 1890, Bülau[15] had advocated drainage of empyema by the "closed" catheter technique, and the relative merits of closed drainage and open drainage by rib resection had been widely discussed in German-speaking countries. Emphasis, however, had been placed on the prevention of retraction of the lung and the quick healing which might be obtained, not on the immediate hazards of open pneumothorax nor on the reduction of functional lung volume to a point below tidal air requirements merely by the admission of air to the pleural space.

In December, 1918, Evarts Graham[16] wrote:

> "During the past year the idea has become prevalent in this country that operations upon the thorax which permit of the free entrance and exit of air into the pleural cavity during the operation can be performed with about the same impunity as abdominal operations. In fact, the belief has arisen that surgeons at the front have found that alterations of the intrathoracic pressure by the admission of air through extensive incisions or large gaping wounds are in themselves not dangerous, and a feeling of optimism exists that now bold surgical intervention on the thorax may be carried out. It has been said consequently that the danger from creating an open pneumothorax in early operations for empyema with permanent drainage is negligible."

Despite the developments and inventions and increased understanding of three and a half centuries, the army surgeons of 1918 were caught off balance when confronted by the old dilemma of Paré. The same problem presented itself in a slightly different form: whether to accept an open pneumothorax with the danger that the vital spirits would depart and vanish or to strive for a closed chest cavity with its hazards of infection and increasing encroachment on pulmonary ventilation by fluid and air in the pleural space. The problem was solved in the same ways that were devised by those who followed Paré—the insertion of a catheter and evacuation of the pleural cavity by "closed *drainage"* (*Bulau*), *or repeated aspiration with needle* and syringe until pleural adhesions localized an abscess that could be drained by rib resection without retraction of the lung In the words of Guthrie, the first object became that of saving life, after that if time were given, the next was to relieve the loaded cavity

This display of ignorance of the principles that form the foundation of thoracic surgery annoyed and dismayed the few surgeons in this country who were familiar with the progress which had been made in Germany prior to the 1914-1918 War. In his Presidential Address of 1922,[17] Samuel Robinson spoke scornfully of those who would apply their war experience to that bugaboo of civilian surgery—bronchiectasis

> "It has always been my belief that the greatest triumph in thoracic surgery will be the surgical eradication of this deplorable disease. The enthusiast returns from the war who has often dragged a lung lobe into a spread thoracic wound, opened it, scraped it, washed it—yea, even removed it—and concluded, therefrom, that intrathoracic surgery is freed of its supposed dangers, and that the possibilities therein are comparable to those in the abdomen. Let him attempt the same performance in his home hospital in a case of lower lobe bronchiectasis Then will he learn what real thoracic pathology means."

There follows a vivid word picture of the surgical undertaking of lobectomy which ends abruptly with the ominous and cryptic statement "Suddenly, it is obviously time to return the patient to his bed Not much has been accomplished."

Samuel Robinson,[18] an advocate of *uberdruck*, had introduced the positive pressure cabinet for thoracic operations at the Massachusetts General Hospital in 1909. Dr. Willy Meyer[19] worked with a chamber at the German Hospital in this city in the same year This universal chamber combined the principles of *uberdruck* and *unterdruck* and allowed for a wide range of pressure conditions. Both Robinson and Meyer were scholars of the surgical literature and both were in close touch with the progress in Germany.

It was against this background that your association which is tonight celebrating its fortieth anniversary was organized By the article of its Constitution which appears as the motto of this address, this Society has brought light to the surgeons of twentieth century America as did John of Vigo and Paré to their colleagues of the Renaissance.

Much of the subject matter of this address was presented in the Mitchell Banks Lecture at the University of Liverpool on November 3, 1948, but not published.

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A NEW CLASSIFICATION FOR COARCTATIONS OF THE AORTA

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SINCE the first case report of a coarctation of the aorta was presented by Paris[1] in 1791, no adequate system or method of classification has been devised. Bonnet,[2] a French pathologist, in 1903 described the present commonly used system of classification of these lesions calling one an "adult" type and the other and "infantile" type. Most writers[3-7] on this subject have criticized this system, usually for more than one reason. In general the reasons have been the incompleteness of the system, the omission of the rarer forms of the anomaly, no indication of [illegible] te meaning denoted relative to the anatomy, pathology, or pathologic [illegible] of the particular situation.

We agree with the [illegible] on the subject so well phrased by these writers. Bonnet's us[illegible] "adult" and "infantile" is misleading. An adult may have th[illegible] riety or an infant may have the "adult" form. Burford [illegible] ation of the problem when he suggested classifying these [illegible] of the location and patency of the ductus arteriosus. Th[illegible] improvement from the physiologic standpoint, but still [illegible] desired. A similar situation prevails with the terms Bah[illegible] advocated for these lesions. He called one type "uncom[illegible] the others "complicated coarctation." The classifica[illegible] attempted to encompass the various possibilities but [illegible] prominent anatomic lesions while accentuating less[illegible] anomalies or pathologic findings.

The lack of [illegible] understanding and absence of unanimity of opinion as to the underly[illegible] ctors concern[illegible] with the causation of the condition and the mechanism of [illegible] lopment of aortic coarctation provide no etiological leads to a fully accept[illegible] classification. Necessarily, recourse must be had to those anatomic and [illegible] ysiologic features which permit clinical differentiation of the several variant[illegible] this lesion.

In response to the clearly defined need for a completely new method of classification of these lesions one of us (W. M. L.) has devised a system which we believe to be superior to any evolved thus far, and one which probably is acceptable clinically. This system was designed from the standpoint that the coarctation is the primary lesion but gives consideration to the existence of intimately associated anomalies. Remotely associated congenital malformations and those occurring elsewhere are excluded by definition.

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TABLE I. CLASSIFICATION FOR COARCTATIONS OF THE AORTA

Type I	Coarctation of the aorta with	—ligament (ligamentum arteriosum)
Type II	Coarctation of the aorta with	—shunt, proximal or distal (patent ductus arteriosus
Type III	Coarctation of the aorta with	—hypoplasia
Type IV	Coarctation of the aorta with	—(isthmus) interruption
Type V	Coarctation of the aorta with	—site elsewhere (specify) a. ascending aorta b. aortic arch c. descending aorta (thoracic or abdominal) d. multiple coarctations

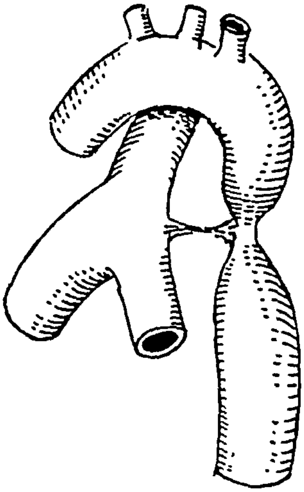

Fig. 1.—Type I. Coarctation of the aorta with ligament (ligamentum arteriosum).

Type I. *Coarctation of the Aorta With Ligament.*—(Fig. 1.) According to Gross[4] this type encompasses fully 98 per cent of all coarctations of the aorta. This is the common type of lesion which is encountered just distal to the origin of the left subclavian artery in the course of the descending aorta. "With ligament" indicates that the ductus arteriosus has become obliterated and no longer is patent or capable of acting as a shunt. This being the case no importance can or should be attached to the point of attachment of this embryonic remnant to the great vessel. Its chief importance to the surgeon is as an aid in locating and dissecting out the left recurrent laryngeal nerve. These lesions usually involve but a very short segment of the aorta. However, those lesions which involve a longer segment but still occur in this region are included under this heading.

Type II. *Coarctation of the Aorta With Shunt, Proximal or Distal (Patent Ductus Arteriosus).*—(Fig. 2, *A* and *B*.) The location of the lesion under this heading is approximately the same as in Type I, but there are very important differences.

Whether the ductus is proximal or distal to the coarcted segment is significant. Those occurring proximal provide a passage for a left-to-right shunt of blood as a logical result of the application of the higher arterial pressure in the aorta against the lower pulmonary arterial pressure. However, if the ductus arteriosus joins the aorta below the point of the aortic narrowing the higher pulmonary artery pressure tends to produce a right to left shunt into the lower aortic segment. Thus, the lower portion of the body may be nourished largely by venous blood from the pulmonary artery. It is in these cases of right to left shunt that an observer may note cyanosis limited to that portion of the body which is below the crest of the ilium.

If the shunt is measured by catheterization and calculated in liters per minute, a number may be placed after the term "proximal" or "distal" to indicate the amount of the shunt.

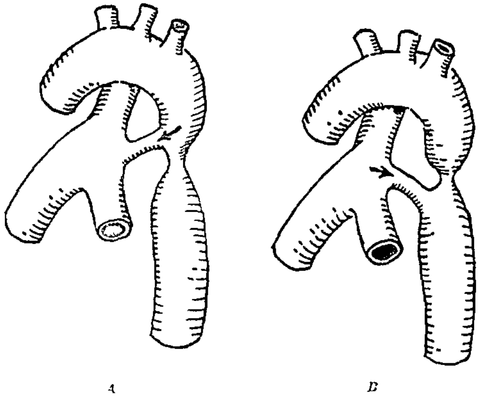

Fig. 2.—Type II (coarctation of the aorta with shunt), proximal or distal (patent ductus arteriosus).

Theoretically there are two remaining situations which may occur in this type of anomaly which must be considered for the system to be complete. If for any reason the shunt should reverse itself from its usual (foregoing) course, the word "reversal" may be used followed by a number which indicates the amount of flow. Lastly there is a possibility that the shunt, when measured, may be in "balance." To give an example:

Type II (coarctation of the aorta with shunt), proximal, 1 L./min.

or

Type II, distal, 3 L./min.

or

Type II, proximal, reversed, 1.5 L./min.

or

Type II, distal, reversed, 2 L./min.

or

Type II, distal, balanced.

The various combinations of possibilities readily come to mind and the ease and simplicity with which they may be identified are apparent.

Type III. *Coarctation of the Aorta With Hypoplasia.*—(Fig. 3.) In hypoplasia, the aorta in most instances is of normal size up to the site of the pathologic lesion. Distal to it, the entire length of the great vessel is grossly diminished in size usually including its bifurcation and the iliac arteries. Once the involved coarcted segment, which usually is found at the site of Type I lesions and not long, has been excised and continuity re-established by an adequate anastomosis, the hypoplastic portion may prove to be an adequate passageway for blood flow. Associated anomalies of the left subclavian artery or an anomalous origin of the innominate or left common carotid artery may give rise to very unusual and interesting physical findings and radiologic observations, but have little to do with the classification of the basic lesions. Rarely, the hypoplasia may involve the aorta from its origin and include the coarctation.

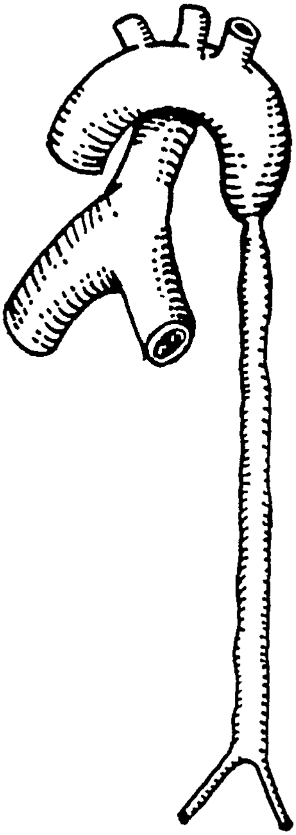

Fig. 3.—Type III. Coarctation of the aorta with hypoplasia.

We do not accept the idea that the lower pressure in the distal segment in itself is responsible for the small caliber of these uncommon cases. If the lower pressure and a decreased blood flow were responsible in them, hypoplasia also should be a most frequent finding in conjunction with Type I lesions.

Type IV. *Coarctation of the Aorta With Isthmus Interruption.*—(Fig. 4.) The isthmus of the aorta is that portion which lies just distal to the origin of the left subclavian artery and before the beginning of the descending thoracic portion of the artery. In the relatively long gap between the proximal and distal segments of the interrupted aorta there is, in most instances, a rudimentary remnant represented by a small fibrous strand of tissue which joins the two portions. Less commonly, this band may be lacking.

Invariably, in this condition, there is a large patent ductus arteriosus arising from the usual site on the pulmonary artery and inserting into the beginning of the distal segment of the aorta. Through this ductus, venous blood enters the lower segment to nourish the lower part of the body. Hence, cyanosis exists below the level of the iliac crests. These cases must be separated clinically from those of Type II with a distal shunt.

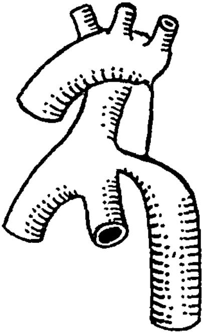

Fig. 4.—Type IV. Coarctation of the aorta with isthmus interruption.

Pathologically, Type IV coarctations may occur without the association of a patent ductus arteriosus, but this is an extremely uncommon form of the anomaly. We have seen only one instance of this situation and it was accompanied by multiple congenital anomalies which caused death soon after birth.

Type V. *Coarctation of the Aorta With Site Elsewhere.*—(Fig. 5.) By definition, this category includes all coarctations located other than at the common site and not specified above. There are only a few such case reports in the literature covering this type, but for the sake of completeness a proper classification must include them. Of all types, this variety is the least frequent.

a. Ascending aorta—Minor degrees of the lesion at this site are compatible with life, but a major degree of involvement may be fatal soon after birth. (Fig. 5, *A.*)

b. Aortic arch—The lesion may occur in this location between the origins of two of the vessels arising from the arch. (Fig. 5, *B.*)

c. Descending aorta—All lesions other than those of hypoplasia are included here that are not Type I, but which occur in the remaining portions of the thoracic or abdominal aorta.

d. Multiple lesions—Various combination of more than one of the above situations may occur. (Fig. 5, *C*.)

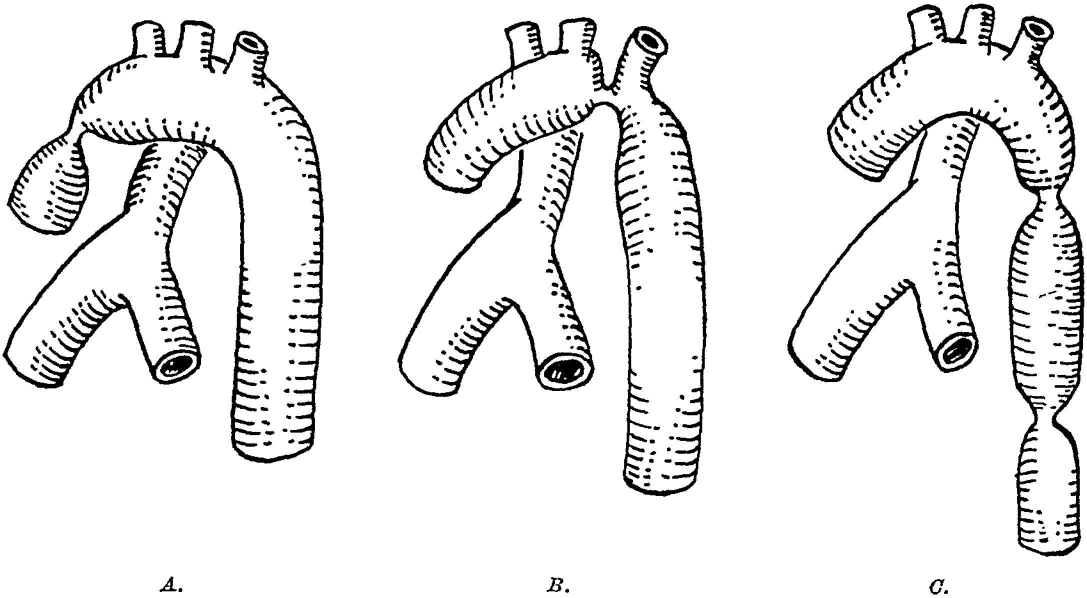

Fig. 5.—Type V. Coarctation of the aorta with site elsewhere. *A*, Ascending aorta, minor degrees. *B*, Aortic arch. *C*, Multiple lesions.

DISCUSSION

A definite need for a comprehensive yet simple classification of coarctations of the aorta has been attested to in the writings of numerous authors. This need has been magnified by the developments of the current era of surgery. Viewed in the light of its magnitude, the surgical treatment of these anomalies is well defined and established with a very high cure rate and a very low mortality and morbidity rate. It seems self-evident that the cardiovascular surgeons of the world should have a standard, generally accepted, system of designation and classification of these anomalies so that writings, teachings, and communications on the subject will be facilitated.

In the foregoing classification, an attempt has been made to list the lesions by type in the order of their comparative incidence, the most common type being placed first and the rarest last.

It is fitting to explain why the isthmus interruption type (Type IV) with its patent ductus arteriosus should be differentiated from the Type II *shunt, distal*. The only feature common to these lesions is the patency of the ductus arteriosus producing a right-to-left shunt to the lower aortic segment and venous perfusion of the portion of the arterial bed. Here the similarity ends. If and when the etiology of these anomalies becomes known, we venture to say that the two types under discussion will vary appreciably as to their origin. It seems to us that the importance of physical absence of a segment of the aorta is of such anatomic, pathologic, and physiologic import as to justify a distinct and separate designation.

The fact that in nearly every Type IV patient either an aortic homograft or a plastic prosthesis will be required to bridge the relatively long defect, whereas simple resection of the coarctation and direct end-to-end anastomosis will suffice in the vast majority of Type II lesions serves further to emphasize their dissimilarity.

SUMMARY AND CONCLUSIONS

The need for a more satisfactory method of classifying coarctations of the aorta has been noted. A brief review of the current confusion and the incompleteness of prevailing methods has been presented.

A completely new and comprehensive, but short and simple system has been devised for their classification and presented with a brief discussion of its propriety.

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BACTERIAL AND MYCOTIC ENDOCARDITIS FOLLOWING CARDIAC SURGERY

Report of a Case

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BACTERIAL endocarditis is now recognized as an occasional complication of cardiac surgery. A review of the literature has revealed a single case of *Candida albicans* endocarditis following cardiac surgery. We report here a case of bacterial and mycotic endocarditis following circumferential suture of the mitral valve ring.

CASE REPORT

A. H., a 32-year-old white male Baptist minister, was admitted to the Cardio-thoracic Surgical Service (Robert P. Glover, M.D., Chief) of The Presbyterian Hospital in Philadelphia on Sept. 1, 1955. His chief complaint was a "rattle on chest" of one year's duration. A heart murmur was noted during an army physical examination in 1942 and, in 1945, he complained of "palpitations." His general condition was good until one year prior to admission when he developed a "rattle on chest," which was relieved in the erect position. He remained active, however, on digitalis and diuretic therapy. He had no peripheral edema and no cough. No history of rheumatic fever could be obtained.

His past medical history included the usual childhood diseases, a tonsillectomy and pneumonia in childhood, and a left nephrectomy at 17 years of age for a "pus kidney." One sister had died of rheumatic fever.

On admission he was a well-developed, well-nourished, white man in no acute distress. The blood pressure was 120/75 mm. Hg and pulse rate 68 per minute. The point of maximal cardiac impulse was felt at the anterior axillary line in the sixth intercostal space. A normal sinus rhythm was present. A Grade 3 "harsh" systolic murmur was transmitted throughout the entire precordium, and a Grade 2 systolic thrill was present at the apex. A Grade 2 "rough" systolic murmur was also present in the aortic area. No diastolic murmurs could be heard. The diagnosis on admission was rheumatic heart disease with predominant mitral insufficiency and associated aortic stenosis. The patient was regarded as a good operative risk.

The results of initial studies were as follows: hemoglobin 15.8 Gm. per 100 ml.; erythrocytes 5,700,000; leukocytes 7,500; normal differential; urinalysis negative; sedimentation rate 0 mm. per hour; bleeding time 1 min., 45 sec.; coagulation time (capillary method) 4 min.; prothrombin time 12.5 sec. (100 per cent). The electrocardiogram was reported as showing combined heart strain and probable atrial hypertrophy. The chest x-rays were reported as compatible with mitral insufficiency and some degree of mitral stenosis.

An attempt at cardiac catheterization was not entirely satisfactory because of venospasm. The right saphenous vein eventually had to be used and the pulmonary artery

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From the Laboratories of The Presbyterian Hospital in Philadelphia, Pa.

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could not be entered The incomplete study revealed a moderate elevation of right ventricular pressure and a normal right atrial pressure An interventricular septal defect was thereby ruled out

With a preoperative diagnosis of predominant mitral insufficiency, operation was undertaken on the eighth of September A total circumferential suture of the mitral ring[1] was accomplished without apparent difficulty The three hour operative procedure was well tolerated by the patient He received 1,500 c c of whole blood while on the operating table At operation, the left atrium and ventricle were moderately enlarged and the right atrium and ventricle were enlarged to a lesser degree A Grade 4 systolic thrill was felt over the left atrium and pulmonary veins The aortic valve seemed free of disease after examination by external palpation No thrombotic material was present in the left auricular appendage On internal palpation of the mitral valve, a 5 finger annulus with a 3 finger valve orifice was found The mitral leaflets were thin and pliable but did not approximate in systole With the purse string in place the annulus was reduced to a 3 finger diameter with favorable pressure changes The insufficiency was thought to be improved by 90 per cent

The immediate postoperative course was uneventful A few râles in the chest on the fifth day cleared after Thiomerin was given On the twelfth postoperative day, a chill and fever of 104° F developed *E coli* was recovered from the urine and hemolytic *Staphylococcus aureus* from the blood A diagnosis of acute bacterial endocarditis was made, and treatment with intravenous penicillin was begun For eight days the temperature spiked between 99° and 104° F

From the fifth to the eighth week of hospitalization, the patient's condition remained stable, and the temperature never rose above 98 6° F Intravenous penicillin was continued for eight weeks, along with other antibiotics (Fig 1), and then was supplanted by oral penicillin In less than twenty four hours, chills and fever of 102° F recurred, and a hemolytic *Staphylococcus aureus* was again recovered from the blood Intravenous penicillin was restarted in larger doses, and at the end of the ninth week the patient's temperature was again normal Some abdominal pain and tenderness, nausea, and tinnitus were present but disappeared when aspirin was discontinued

From the tenth to the thirteenth week the patient's temperature remained below 99 4° F, although his general condition remained poor He appeared weak and lethargic, with occasional bouts of acute pulmonary edema During the eleventh week, adrenal cortical insufficiency was suspected, and serum electrolyte studies were as follows Na 121 meq /L, K 5 9 meq /L, Cl 78 8 meq /L, and CO 15 7 meq /L The urinary 17 ketosteroid excretion was 2 3 mg per twenty four hours With hormone therapy, including ACTH, cortisone, and DOCA (desoxycorticosterone acetate), and adequate intravenous electrolyte solutions, the patient's strength and appetite improved

During the thirteenth week in the hospital, the intravenous penicillin was once more discontinued in favor of oral penicillin Within thirty six hours, the patient's temperature began spiking again and varied between 97° and 104 4° F until death Hemolytic *Staphylococcus aureus* was once more recovered from the blood during this period It was suggested that a misplaced "purse string" might be harboring the organism, in which case the prognosis would seem hopeless Intravenous penicillin was restarted, but this time without much success A serum calcium of 8 2 mg per 100 ml was recorded following the appearance of physical signs suggesting tetany Intravenous calcium gluconate relieved this situation and elevated the serum calcium to 10 2 mg per 100 ml The patient eventually became extremely lethargic and died 111 days after admission to the hospital

Autopsy Findings—At autopsy a diffuse fibrinous pericarditis was found, the pericardium being loosely adherent to the shaggy epicardial surface The pleurae showed areas of fibrous adhesions, and the right thoracic cavity contained a liter of pale yellow fluid There was no evidence of arteriosclerosis in the aorta or peripheral vessels The heart weighed 775 Gm and showed marked hypertrophy of the myocardium All cardiac chambers were dilated. The left auricular appendage was absent There was evidence of healed rheumatic valvulitis involving mainly the mitral valve, but also the aortic and tricuspid valves. The mitral ring was widely open, admitting 4 fingers easily and measuring 12.0 cm. in circumference. The mitral cusps were markedly fibrosed and contained deposits of calcium. The chordae tendineae were exceedingly thick and firm. The posterior hemicircumference of

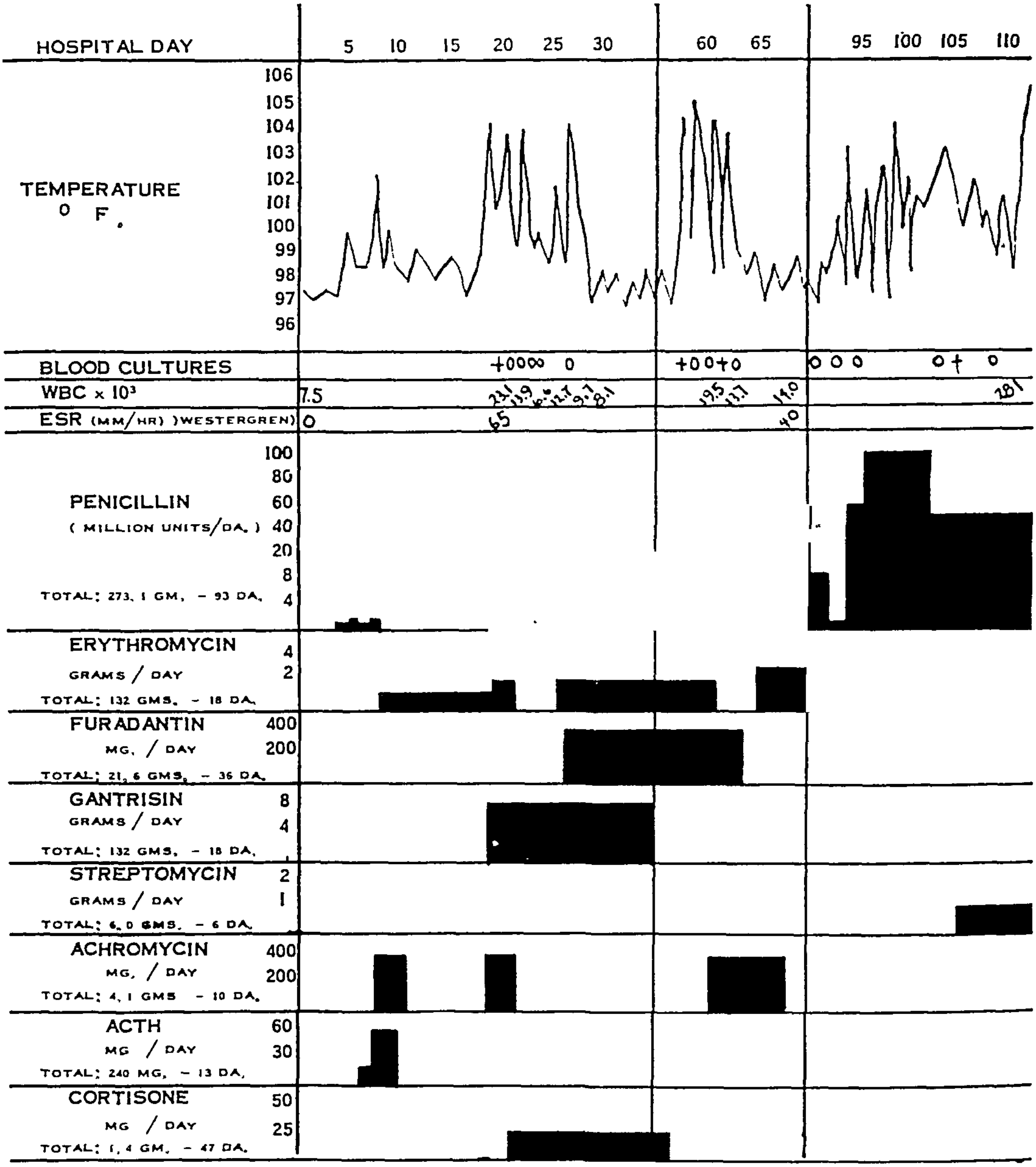

Fig. 1.—Clinical course of patient with deletion of two stable periods.

the "purse-string" suture was seen free in the left auriculoventricular lumen. This portion of the suture measured 6 cm. in length and was covered with a 5 by 2 by 2 cm. irregular, friable, grayish-brown to brownish-red thrombus (Fig. 2). This thrombus was made up of fibrinous material containing colonies of Staphylococci and extensive fungus growth. Shallow ulcerations covered with fibrinous material were seen at the sites where the suture was attached to the wall of the heart. Microscopic examinations of the heart revealed myocardial hypertrophy, small focal infarcts, focal fibrosis, fat replacement, hemosiderosis, and foreign body granulomata. Evidence of healed rheumatic valvulitis and reactivated pancarditis was also observed. The areas of ulceration displayed acute and chronic inflammation without bacterial or fungus growth.

The lungs together weighed 1,475 Gm. and showed severe congestion and edema. The spleen weighed 700 Gm. Marked passive congestion and multiple infarcts were noted A small artery exhibited necrotizing vasculitis, apparently resulting from bacteremia. The left kidney was absent, having been removed previously. The right kidney weighed 400 Gm. and showed compensatory hypertrophy. Several recent infarcts and areas of acute and chronic pyelonephritis were observed, although no cocci or fungi were seen. The liver weighed 2,600 Gm. and showed marked chronic passive congestion. Other findings included moderate depletion of the adrenal cortical lipids, diffuse reactive hyperplasia of the bone marrow, and congestion and slight interstitial inflammation of the pancreas.

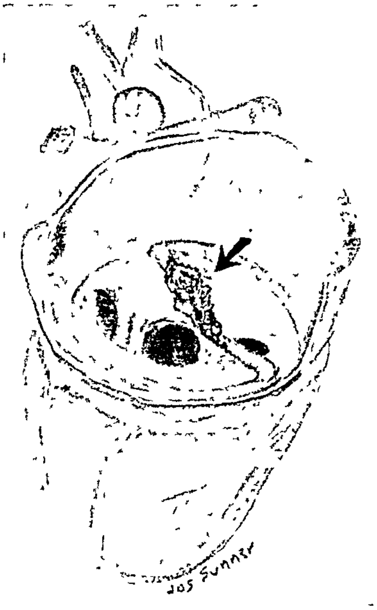

Fig 2 —View of mitral ring showing intraluminal portion of "purse-string" with thrombus

DISCUSSION

Fourteen cases of bacterial endocarditis following cardiac surgery have been reported in the literature.[2-8] Thus, while not a common complication, it occurs with sufficient frequency that it must be regarded as one of the hazards of such surgery. In the majority of instances, the offending organism has been a hemolytic staphylococcus.

Mycotic endocarditis is still a rare disease. Kunstadter and associates,[9] in 1952, found 24 cases in the literature and added another due to *Candida albicans.* Several other well-documented reports have appeared in the past five years.[10-15] Recently a case of mycotic endocarditis following cardiac surgery has been reported.[21] Candida appears to be the most frequent offender, having been responsible for endocarditis in 10 out of 25 cases analyzed by Kunstadter and co-workers.[9] Aspergillus,[10-14] Actinomyces,[11, 13, 15] and histoplasmosis[22, 23] have also been reported as the cause of endocardial involvement.

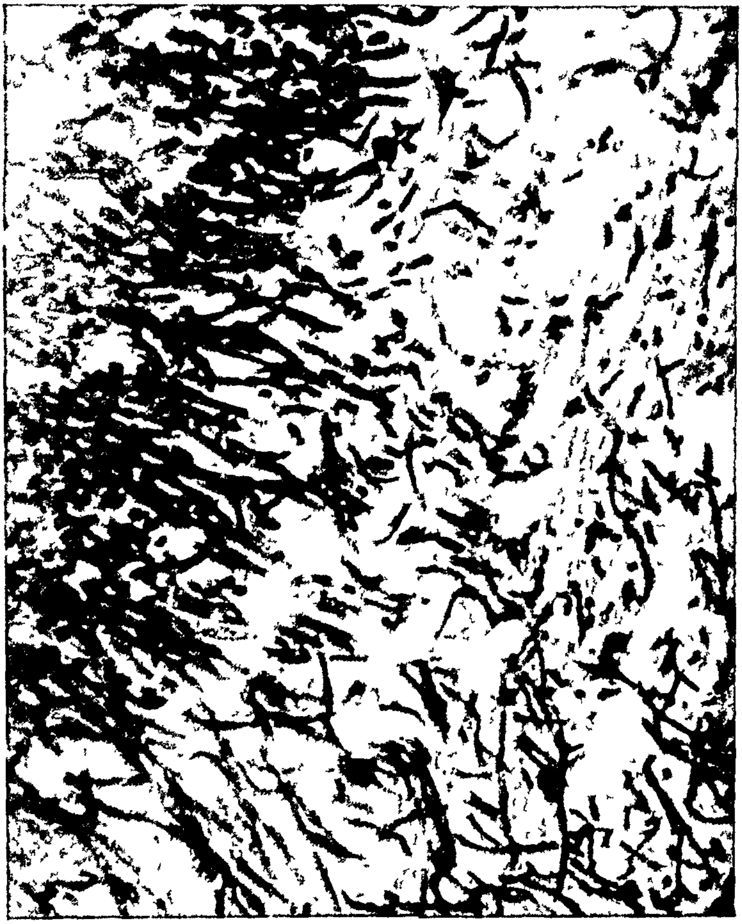

Fig. 3.—Photomicrograph of thrombotic material from "purse-string" showing mycotic growth (periodic acid-Schiff stain)

Endocarditis due to a combination of bacteria and fungi has been previously reported by Joachim,[16] Geiger,[17] Koehlmeier[11] and their associates, but to our knowledge this is the first such report subsequent to surgery of the heart.

At autopsy, a portion of the circumferential suture used to effect reduction in size of the mitral annulus was found lying free in the left auriculoventricular lumen, having been misplaced at the time of surgery. Its presence in the lumen afforded a site for formation of a thrombus in which organisms could lodge and multiply. A hemolytic *Staphylococcus aureus* was recovered from 4 of 17 antemortem blood cultures, and microscopically the thrombotic material from the "purse-string" contained colonies of Staphylococci. In addition, this thrombotic material also showed extensive mycotic growth. The fungi, identified as *Candida albicans* with hematoxylin and eosin and periodic acid-Shiff stains,* were arranged in a foliate pattern, showing septate mycelia and yeastlike cells (Fig. 3). Since the mycotic component was entirely unsuspected at the time of autopsy, culture studies were not done.

The extensive use of antibiotics and chemotherapeutic agents in recent years apparently has resulted in an increased incidence of mycotic infection,[9, 12, 18, 19] as predicted by Zimmerman in 1950.[14] Evidence has also been presented that widespread use of steroid hormones may be responsible for enhancement of fungous infections.[18, 19, 20] In addition, increased susceptibility due to the primary disease and the nature of the local lesion is also important.[10, 19] In the case presented all of these conditions probably obtained in favoring growth of the fungus.

While differences between sensitivity of organisms to antibiotics in vitro and in vivo are well recognized, it is of interest here that this Staphylococcus was penicillin resistant in vitro, yet seemed to be controlled by large doses of intravenous penicillin. Although other antibiotics were used sporadically, chills and fever with positive blood cultures promptly recurred on the two occasions when intravenous penicillin was discontinued in favor of smaller doses of oral penicillin.

SUMMARY

A case of bacterial and mycotic endocarditis following circumferential suture of the mitral ring is reported. Its unusual features and the probable contributory factors are discussed.

We are indebted to Dr. Robert P. Glover and his associates for permission to use this material and to Mr. Joseph Sunner for the illustrations and photograph.

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*The organism was identified morphologically by Dr. Dale Schultz, Assistant Professor of Pathology, University of Indiana.

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EXPERIMENTAL BILATERAL TRANSSTERNAL THORACOTOMY: FACTORS IMPROVING SURVIVAL

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INTRODUCTION

A BILATERAL transsternal thoracotomy affords optimal access to the heart and great vessels, and is of particular value in experimental surgery because it simulates the technical exposure employed in human patients during most intracardiac and pericardiac operations. Earlier, this approach was attended by a high mortality in our laboratory, and many visitors to our department stated that they had abandoned this incision in their own experimental preparations in favor of the less traumatic unilateral thoracotomy, because of inability to obtain long-term survival following the more extensive procedure.

In conjunction with our recent efforts to prevent ventricular fibrillation during right ventriculotomy and hypothermia, an experimental operative technique which reproduced its clinical counterpart was considered essential. This required the bilateral transsternal exposure, and stimulated us to evolve a program of operative management and postoperative care which would yield an acceptable mortality rate.

MATERIALS AND METHODS

Mongrel dogs weighing from 6.4 to 16 3 Kg. were chosen at random from a large isolation run. The only selection involved was an attempt to exclude those animals which were obviously ill or within the extremes of age. None received any special preoperative diet or therapy. No pre-anesthetic medication was employed. Sodium pentobarbital was administered intravenously as the anesthetic. The dosage was adjusted to eliminate the lateral canthal reflex with preservation of the medial, which required about 30 mg. per kilogram of body weight. Following tracheal intubation, the animals were placed in an ice-water bath, and positive-pressure respiration was begun with 100 per cent oxygen, at about 40 respirations per minute. Removal from the ice water was done when a rectal temperature of 29° C was reached. A further decline of 3° to 5° C. occurred, so that the experimental procedures were performed at a rectal temperature of 24° to 26° C.

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All of the animals underwent bilateral transsternal thoracotomy through the fourth intercostal space. Right ventriculotomy was then performed during an inflow-outflow circulatory occlusion of from 8.5 to 13.0 minutes.

After the release of the inflow occlusion and reconstitution of a normal circulation, the following protocol was observed carefully:

1. Continued observance of aseptic technique.
2. Loose closure of the pericardium with interrupted sutures of No. 4-0 black silk.
3. Bilateral water-sealed chest suction for 2 to 8 hours, depending on the immediate postoperative activity of the animal.
4. Approximation of the sternum with two crossed parasternal No. 28 or No. 32 wires.
5. Approximation of the rib cage on either side with a single No. 28 or No. 32 paracostal wire.
6. Careful four-layer (intercostal bundle, pectoral muscles, fascia, and skin) closure of the thorax.
7. Replacement of blood loss with whole blood or a plasma-volume expander—about 10 to 15 ml./Kg.
8. Intramuscular administration of Metrazol (pentylenetetrazol) 100 mg. (1 ampule) per kilogram of body weight, immediately postoperative.
9. Rewarming in a water bath at 40° to 45° C., where the animal remained until a rectal temperature of 32° to 36° C. was achieved (spontaneous respiration always followed cessation of the positive-pressure breathing in this temperature range).
10. Routine bronchoscopy on the morning following operation of all dogs weighing 10 Kg. or less, and immediate bronchoscopy of all other dogs developing persistent cough on the first or second postoperative day, small doses of sodium thiopental being used intravenously as the anesthetic.
11. Administration of antibiotics (penicillin and streptomycin) for the first 72 postoperative hours.

If ventricular fibrillation occurred at any point during the operative procedure, it was allowed to persist throughout the predetermined period of circulatory occlusion. Then electrical ventricular defibrillation was achieved as described elsewhere, with particular attention to the prevention of epicardial and myocardial burns.[1]

"Survival" has been assigned arbitrarily as implying "alive and well" seven days postoperative.

RESULTS

Thirty successive dogs underwent bilateral transsternal thoracotomy, hypothermia, and right ventriculotomy as described. Of these, 10 developed ventricular fibrillation during the operative procedure, and each was successfully defibrillated electrically. Twenty-nine animals survived. The one death occurred as a result of an overdose of barbiturate prior to bronchoscopy on the first postoperative day (Table I).

TABLE I. SURVIVAL OF DOGS UNDERGOING BILATERAL TRANSSTERNAL THORACOTOMY DURING HYPOTHERMIA

DOGS UNDERGOING TRANSSTERNAL THORACOTOMY	ALIVE 7 DAYS POSTOPERATIVE	SURVIVAL
30	29*	97%

*The one death was due to an overdose of barbiturate prior to bronchoscopy.

DISCUSSION

The protocol of thoracic closure and early postoperative care as presented here permits the use of the bilateral transsternal thoracotomy in experimental animals without imposing an increased operative hazard. The relative importance of each component has not been evaluated, but presumably no single aspect is crucial.

Closure of the pericardium assures a stable midline position of the heart; otherwise, torsion of the venae cavae could occur with resultant embarrassment of right heart filling when the animal is turned from side to side during the recovery period.

Careful examination of the thorax, followed by underwater drainage of both hemithoraces, permits complete and permanent expansion of the lungs. Meticulous suture of the posterior angles of the incision as well as the parasternal regions is essential.

Maintenance of a normal blood volume appears as practical here as in other major operative procedures.

Rapid rewarming and reversal of barbiturate effect with Metrazol allows the animal to be awake and ambulatory a few hours following operation. In addition, Metrazol encourages deep breathing with its attendant benefits. No convulsions have been noted following the use of this analeptic in the dosage employed here. No clinical evidence of heart failure (tachycardia, râles, or distention of tongue veins) was observed during or after the period of rapid rewarming to a rectal temperature of 33° to 38° C.

On the morning following operation, bronchoscopy was performed in most animals. In those that underwent endoscopy, mucous plugs of varying sizes were recovered from the tracheobroncial tree. A complete cast of the lower trachea and both bronchial trees was removed from one animal. The formation of these accumulations of mucus is thought to be secondary to poor tussive efforts in the early postoperative period, and serves as a further recommendation for the expeditious return of the animal to consciousness postoperatively.

In addition to the technical advantage of the transsternal thoracotomy, a more subtle indication for its use in cardiovascular research may exist.

Hypothermia has proved to offer a satisfactory means of correcting certain cardiovascular anomalies under direct vision, i.e., interatrial septal defect, pulmonary stenosis, and aortic stenosis.[2-5] However, operating within the open ventricles during hypothermia has had limited application because of the threat of ventricular fibrillation. Recently, efforts have been made in many laboratories to overcome this disadvantage.[6-10] However, the basic experimental preparation varies from one investigating group to another. Our own experience in the past two years suggests that the bilateral thoracotomy is associated with a higher incidence of ventricular fibrillation during hypothermia than is the unilateral approach. If this is indeed so, standardization of this fundamental aspect of the experimental preparation among research groups would seem advisable.

The standard procedure of choice may be the bilateral approach, since it duplicates the clinical situation under which many of the experimental techniques will eventually stand trial.

SUMMARY AND CONCLUSIONS

Bilateral transsternal thoracotomy provides optimal exposure of the heart and great vessels. It is of particular value in experimental cardiac surgery, because it duplicates the clinical situation and provides a standard preparation for interlaboratory comparisons. A method of wound closure and early postoperative management is presented which permits performance of this more extensive procedure with little or no increase in operative risk.

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PNEUMOMEDIASTINUM PHYSIOLOGIC OBSERVATIONS

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AIR may enter the mediastinum following trauma involving the trachea or esophagus, during operative procedures in the neck in which the pretracheal fascia has been opened, or "spontaneously" from the lung Forbes and co workers[1] have demonstrated experimentally that, following opening of the pretracheal fascia as in a tracheotomy, air may be sucked into the mediastinum to produce mediastinal emphysema and a pneumothorax This process is augmented by labored respiration Bouden and Schweizer,[2] in 1950, reported 18 cases of pneumothorax and 2 cases of mediastinal emphysema following operations upon the head and neck, which developed presumably by this same mechanism

That these patients have severe symptoms, even leading to death, has been reported and confirmed many times Usually it has been assumed that the mechanism producing the symptoms is the development of air under tension in the mediastinum This causes embarrassment of the venous return in the venae cavae, lowered cardiac output with hypotension, and circulatory failure unless the pressure is released As there is some reason to doubt that sufficient pressure can be developed in the mediastinum to impede venous return seriously, this problem has been experimentally reappraised

Jehn and Nissen[3] injected air into the mediastina of puppies and did produce asphyxia with a falling arterial blood pressure leading to peripheral circulatory collapse However, these experiments were not controlled to determine the effects of the frequently accompanying pneumothorax Later, Ballon and Francis[4] injected air into the mediastinum, inflated rubber balloons in the posterior mediastinum, or cut windows in the lower trachea to produce mediastinal emphysema Here again, these studies were not adequately controlled to determine the effects of frequently accompanying pneumothorax Nor does a rubber balloon simulate free air in the mediastinum as the confines of the mediastinum are not as definite as those of the rubber balloon

METHODS

Rabbits and adult mongrel dogs were anesthetized with pentobarbital, and a long needle was introduced through the jugulum passing through the trachea into the visceral space of the upper mediastinum Air was injected slowly

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in graded amounts with constant recording of the pressure in the mediastinal air pocket. Intercostal catheters were inserted bilaterally and attached to water-seal drainage with suction to detect the development of a pneumothorax. Catheters were likewise introduced into the venae cavae, the right atrium, and the femoral artery. Pressures were recorded by mercury manometers and on Sanborn electromanometers using Statham strain gauges.

Four dogs had poudrage of both pleural cavities with 5 per cent iodized talc to produce a sterile pleuritis and complete pleural symphysis. After a period of three to four weeks, these dogs were subjected to the experiment as outlined above to check if significant tension could be produced when the lateral mediastinal fascias were buttressed by the lung.

Rabbits were used in addition to dogs because of the concept that the dog has an instable or flimsy mediastinum. Our anatomic studies demonstrated that, while the anterior mediastinum of the dog is composed only of a sheet of fatty mesentery and is quite mobile and tenuous, the mediastinal fascias surrounding the visceral space are quite tough and have the same anatomic features as that of other experimental animals. In fact, it appears to us that these fascias in the dog are tougher than those of the rabbit. Both experimental animals differ from human beings in that the exposed inferior vena cava projects through the right pleural space covered only by a thin sheet of pleura and completely surrounded by lung while, in man, the inferior cava has relatively a much shorter course in the thorax before entering the right auricle and is much better protected from pleural pressures.

Last, to ascertain whether the experimental results on these animals might be validly transferred to man, the rupturing pressure of the human mediastinum was measured at the autopsy table. After removal of the breast plate, the chest was flooded with water. A long needle was injected through the jugulum into the visceral space of the posterior mediastinum, and air was injected as in the experimental animals with constant pressure recordings.

RESULTS

Our method was universally successful in producing marked mediastinal emphysema in all animals. It was found that the rapid injection of air under high pressure led immediately to a pneumothorax. Accordingly, initial injections of air were made very slowly with constant monitoring to prevent a rise of the pressure above 40 mm. Hg.

In only 1 of 16 normal dogs was any marked effect produced by the pneumomediastinum alone. Dog 10 developed very extensive mediastinal emphysema, subcutaneous emphysema, and an extremely tense pocket of air in the cervical mediastinal visceral space which could be easily seen and felt. During this period, however, while the superior vena caval pressure rose 4 mm. Hg (equivalent to approximately 52 mm. of water pressure), the right atrial pressure showed no change, and the femoral arterial pressure fell only 24 mm. Hg. The inferior caval pressure showed no change. Cervical mediastinotomy on this dog immediately relieved these slight pressure changes and, in addition, helped to decompress the accompanying right pneumothorax. Further air injected into the mediastinum left via the mediastinotomy, preventing further complications from the pneumothorax.

In the remaining 15 dogs, all observable effects were due solely to the accompanying pneumothorax (Fig. 1). This invariably effectively decompressed the mediastinal emphysema but in itself proved fatal if unrelieved. The effects of the tension pneumothorax were easily reversed by aspiration unless there had been too much delay. The typical preterminal response was a marked rise in the arterial, atrial, and vena caval pressures, followed by a rapid decline and death (Fig. 2). This is a characteristic response to progressive acute anoxia, regardless of the cause.

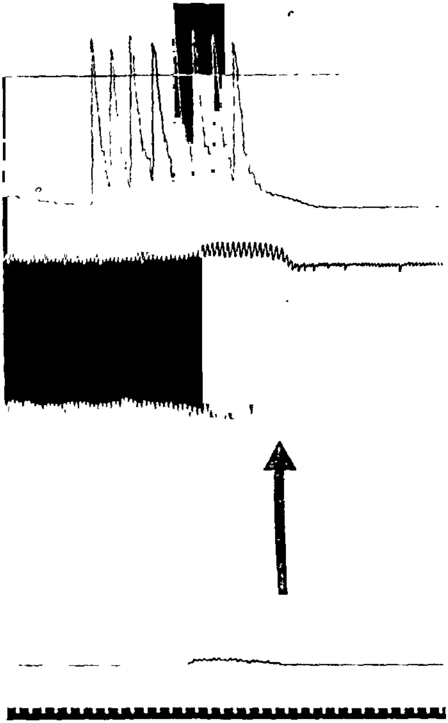

Fig. 1.—Pneumothorax effect. *Top line* = base line. High spiked line reflects the pressures in the mediastinal air pocket, showing the immediate fall of pressure at the conclusion of each 50 c.c. injection. *Next line* = right atrial pressure. *Fourth line* = femoral arterial pressure, and *lower line* shows the inferior vena caval pressure. Time markers represent 5 seconds. At the arrow, the tension pneumothorax was decompressed. Note the return of all measurements to normal.

In addition to rupturing into the pleural cavities, air was found to dissect into the subcutaneous tissues or inferiorly along the fascial planes into the retroperitoneal spaces (Table 1). Usually one of these routes predominated.

TABLE I. SITES OF AIR-DISSECTION

TOTAL	C.A.P.	P.T.		R.P.	S.E.
		RT.	LT.		
16 (Dogs)	5	13	12	1	5
4 (Poudraged Dogs)	4	3	2	4	1
5 (Rabbits)	0	4	4	1	5

Sites of air-dissection noted in experimental animals. C.A.P. = cervical air pocket; P.T. = pneumothorax; R.P. = retroperitoneal; S.E. = subcutaneous emphysema.

Frequently, subcutaneous emphysema was noted first and, once this route of escape was established, several liters of air could be injected rapidly into the posterior mediastinum with no deleterious effects being observed.

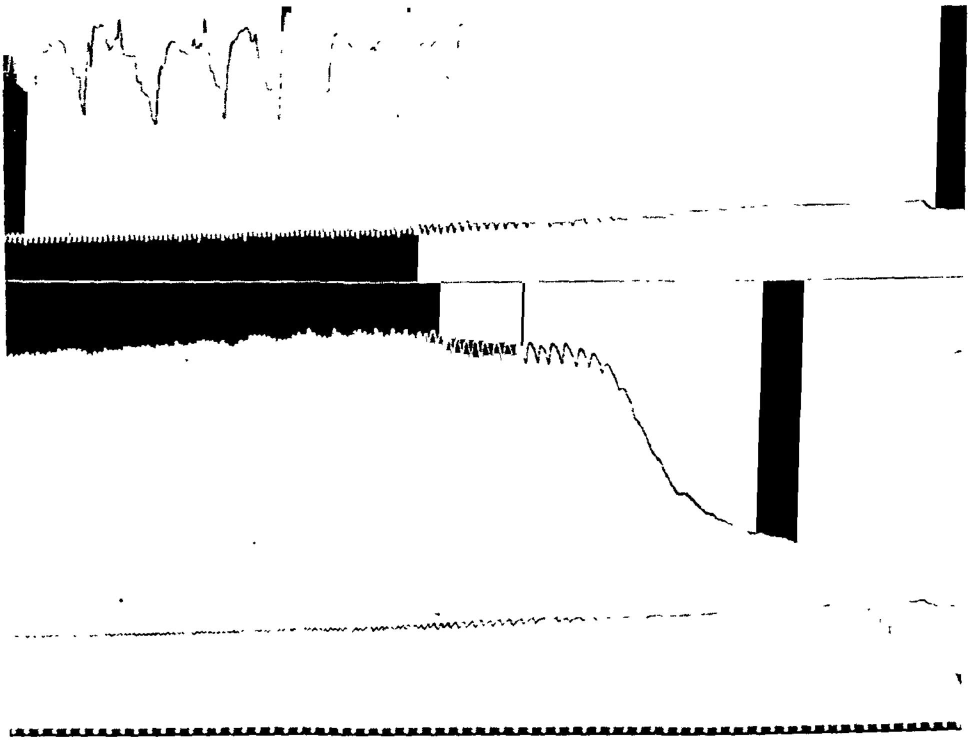

Fig. 2.—Terminal anoxic effect of tension pneumothorax. Tracings are the same as in Fig. 1, except for shift of the base line. Note the rising caval and atrial pressures and the initial rise of the arterial pressure with its terminal fall. Again the mediastinal air pocket pressure is seen to return to normal after each injection.

Similarly, marked mediastinal emphysema was produced in 5 rabbits. No rabbit developed physiologic effects from the mediastinal emphysema alone. The rabbits were much more prone to develop subcutaneous emphysema than the dogs. They likewise developed unilateral or bilateral tension pneumothoraces and showed only the effects of the tension pneumothoraces. Retroperitoneal air was noted in only one of the rabbits.

In the 4 poudraged dogs, only a partial pleural symphysis was obtained. Extensive mediastinal adhesions, however, were noted in each, and the mediastinal pleura appeared to have been effectively strengthened, for these animals developed pneumothoraces late and only after the appearance of extensive subcutaneous and retroperitoneal collections of air. All developed a very tense cervical air pocket within the visceral space.

Effects from mediastinal emphysema alone were noted in 3 of these 4 dogs (Table II). These changes, while definite, did not cause any appreciable circulatory distress and all were transient. Respiratory difficulty was never occasioned by the pneumomediastinum, subcutaneous emphysema, or retroperitoneal air—only by the pneumothorax.

TABLE II. POUDRAGED DOGS—PRESSURE CHANGES (mm. Hg)

DOG	V.C.	R.A.	F.A.	C.A.P.
1	+6	+4	−40	+6
2	+2	0	0	+4
3	0	0	−10	+4
4	0	0	0	+5

Pressure changes noted in poudraged dogs. V.C. = vena cava; R.A. = right atrium; F.A. = femoral artery; C.A.P. = pressure in cervical air pocket of the visceral space.

The deep cervical air pockets could quickly be decompressed by a cervical incision extending through the pretracheal fascia which is the anterior encompassing fascia of this pocket. Similarly, mediastinotomy would prevent a pneumothorax in most cases, but not always, for some dogs were observed to develop a tension pneumothorax even in the presence of an effective mediastinotomy which was bubbling air freely. Dog 13 was noted to have both an extremely tense cervical air pocket in the mediastinum and a right pneumothorax. The attendant severe respiratory and circulatory symptoms were not relieved by mediastinotomy but by decompression of the pneumothorax. Only rarely could the pneumothorax effect be reversed by mediastinotomy.

In the post-mortem observations on 6 human adults, air was injected slowly to keep the pressure below 20 mm. Hg. Again, the pressure immediately dissipated as air dissected diffusely through the mediastinum. In no case could a persistent tension above 5 mm. Hg be maintained. Air always bubbled out into one or both pleural cavities after injection of only a small amount of air —varying from 20 to 400 c.c. The point of rupture was usually anterosuperior, far from the site of injection.

DISCUSSION

Air injected into the mediastina of rabbits, dogs, and human beings does not produce a tension effect as the air escapes by dissecting along the tissue planes or rupturing into the pleural spaces, and the mediastinal pressure quickly falls to insignificant levels. At no time were we able to develop a persistent pressure within the normal mediastinum greater than about 5 mm. Hg. In dogs which have had the mediastinal spaces buttressed by formation of adhesions or symphysis of the mediastinal and visceral pleurae, slightly greater pressures can be developed which may produce mild circulatory symptoms.

Goldberg, Mitchell, and Augrist[5] found that the rupturing pressure of the mediastinum in infant human cadavers is approximately 40 mm. Hg. At this level, the air would rupture into the pleural space. A slight shift in position might close this rent and often a subsequent pneumothorax would appear on the opposite side.

Our results on human adults would suggest that such a pressure can be attained only with the acute injection as the dissection of the air along tissue planes immediately reduced the pressure to less than 5 mm. Hg. In addition, in each observation the mediastinum ruptured with egress of air into the pleural cavity at pressures less than 20 mm. Hg. It is probable that, in the presence of pulmonary disease with pleural thickening, much higher acute pressures may be attained before the pleura ruptures. Nonetheless, the air will dissect subcutaneously and retroperitoneally and, only in most unusual circumstances, develop a persistent high pressure within the confines of the mediastinum.

This work suggests that symptoms due to a tense pneumomediastinum must be rare and that the effects noted are due to an associated pneumothorax. In fact, once a pneumothorax has developed, it appears to be quite impossible to develop sufficient pressure within the mediastinum to cause any degree of compression of the heart or great vessels. The rise in right atrial pressure noted in all dogs with pneumothoraces suggests that the block is in the pulmonary capillaries and not the venae cavae.

Many of the clinical reports such as those of Karns,[6] Sheinfeld,[7] Gaudrault,[8] and Rydell[9] attribute the beneficent effects of emergency cervical mediastinotomies to decompression of massive mediastinal emphysema. They do not present adequate supporting data, however, to show that the symptoms were due to the mediastinal air. In fact, most of the patients presented had accompanying pneumothoraces. Our experimental observations support the contention of Tuttle[10] that the difficulty is usually due to the tension pneumothorax and not the mediastinal emphysema.

The mediastinal emphysema that we have studied is, of course, completely different from malignant interstitial emphysema of the lungs accompanied by mediastinal emphysema. This has been extensively studied by the Macklins,[11] Hamman,[12] and more recently by Fagin and Schwab.[13] In this condition, the air dissects from the alveoli along vascular sheaths and compresses the pulmonary capillaries. It may, in addition, cause air lock of the alveoli, preventing adequate ventilation. Vanderlaan and Maresh[14] have reported one such case of mediastinal emphysema without a pneumothorax. The patient developed subcutaneous emphysema, cyanosis, dyspnea, and pain and became irrational. A collar incision allowed an immediate egress of air with rapid improvement and an uneventful convalescence. This type of case apparently is extremely unusual.

A mediastinotomy can be of value in preventing a pneumothorax or in preventing further accumulation of air in an existing pneumothorax. It is doubtful, however, that the mediastinotomy will decompress an existing pneumothorax.

Hickam and Frazer[25] Oxygen consumption and minute ventilation were determined on a Collins spirometer in the earlier studies and, more recently, by collection in a Douglas bag and analysis on a Pauling oxygen analyzer. Cardiac

TABLE I. PHYSICAL CHARACTERISTICS AND DIAGNOSIS OF PATIENTS WITH CHRONIC PULMONARY DISEASE

CASE	AGE	SEX	S A	V C.	M B C.	EXP. 1 SEC	DIAGNOSIS
A. *Emphysema (Simple)*							
1. J. B.	32	M	1.80	4.0 (82%)	123	47.0%	Localized emphysema
2. G. F.	53	M	1.86	3.7 (80%)	72.2 (66%)	41.8%	Chronic emphysema
3. V. M.	56	M	1.47	2.2 (61%)	39.2 (46%)	39.7%	Chronic emphysema, cor pulmonale
4. G. H.	48	M	1.47	3.1 (83%)	35.1 (38%)	63.0%	Chronic emphysema
5. M. H.	40	M	1.88	2.6 (55%)	34.0 (28%)	38.5%	Chronic emphysema, cor pulmonale
6. H. W.	61	M	1.68	2.0 (47%)	23.7 (25%)	35.4%	Chronic emphysema
7. B. S.	57	M	1.86	1.9 (41%)		25.0%	Chronic emphysema, cor pulmonale
8. M. H.	55	F	1.98	2.3 (59%)	48.1 (53%)	50.0%	Chronic emphysema, cor pulmonale
B. *Emphysema (Complicated)*							
9. L. G.	68	M	1.54	1.6 (42%)	28.8 (36%)	42.0%	Bullous emphysema
10. L. D.	51	M	1.69	1.6 (38%)	39.0 (38%)	74.3%	Bullous emphysema
11. R. H.	42	M	1.87	2.2 (47%)	46.3 (38%)	32.5%	Cystic disease
12. M. H.	40	M	1.50	1.7 (47%)	37.2 (37%)	53.0%	Cystic bronchiectasis
C. *Granulomatous Lung Disease*							
13. V. S.	23	F	1.60	3.2 (80%)	106 (108%)	48.5%	Eosinophilic granuloma
14. D. D.	36	F	1.52	2.0 (66%)	63.7 (77%)	47.5%	Sarcoidosis
15. W. T.	54	M	1.69	2.8 (65%)	37.9 (38%)	34.2%	Silicosis
16. W. B.	47	M	1.79	2.1 (50%)	55 (53%)	50.7%	Granulomatous disease, etiology undetermined
17. C. E.	52	M	1.99	3.0 (60%)	57 (50%)	Total 7 sec.	Cavitary tuberculosis
18. B. L.	73	M	1.58	2.4 (60%)	56.6 (74%)	70.0%	Multiple pulmonary emboli
D. *Carcinoma of Lung*							
19. F. B.	63	M	1.64	2.5 (61%)	78.0 (89%)	55.5%	Primary bronchiogenic carcinoma
20. F. M.	68	M	1.41				Primary bronchiogenic carcinoma
21. G. W.	51	M	1.54	2.0 (51%)	61 (67%)	65.0%	Primary bronchiogenic carcinoma
22. F. B.	49	M	1.67				Primary bronchiogenic carcinoma
E. *Pulmonary Disease With L. V. Failure or Mitral Disease*							
23. E. W.	53	M	1.57				Emphysema, rheumatic heart disease with mitral insufficiency
24. O. B.	66	F	1.57				Periarteritis, tuberculosis
25. E. O.	25	F	1.51	2.1 (70%)	85.0 (94%)	74.0%	Pulmonary and myocardial sarcoid with left ventricular failure
26. R. H.	43	M	1.72	3.7 (86%)	49.8 (45%)	40.0%	Emphysema, rheumatic heart disease with mitral insufficiency
27. W. B.	48	M	2.12	4.2 (80%)	75.0 (54%)	53.7%	Emphysema, bronchiectasis, rheumatic heart disease with mitral insufficiency
28. D. E.	58	M	1.96	3.8 (61%)	57.8 (52%)	44.5%	Emphysema, arteriosclerotic heart disease with mitral insufficiency

S.A. = surface area, V.C. = vital capacity, M.B.C. = maximum breathing capacity; Exp. 1 Sec = per cent of vital capacity expired in first second

UNILATERAL PULMONARY ARTERY OCCLUSION IN MAN

II. Studies in Patients With Chronic Pulmonary Disease

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INTRODUCTION

The technique of unilateral pulmonary artery occlusion has been used in our laboratory in over 100 cases to date. The results in a group of controls have already been reported, with particular reference to flow-pressure relationships.[1, 2] It has been demonstrated that this technique provides a method for increasing unilateral pulmonary blood flow without increasing cardiac output as, for example, by exercise.

It is the purpose of this communication to present the results of studies carried out in a series of patients with various types of chronic pulmonary disease, utilizing the method of temporary unilateral pulmonary artery occlusion.

Standard pulmonary function tests and cardiac catheterization, as well as arterial blood studies, have contributed much to the understanding of the pathophysiology of such diseases.[3, 4] There are still certain problems, however, which remain unsettled. Of particular importance are those patients being considered for resectional lung surgery who are poor risks. In such cases, impaired ventilatory function together with elevated pulmonary arterial pressure has usually been considered a contraindication to such surgery, and the decision to proceed is still empirical.

Recently described work, by Perkins and co-workers,[5] on ventilation-perfusion relationships in the lung and, by Riley and associates,[6-8] on maximal diffusion capacities, promises to aid greatly in evaluating patients with chronic pulmonary disease. Certain results obtained in the present study extend the results by these authors. The clinical application of the method of unilateral occlusion in the study of these parameters in patients with various forms of chronic pulmonary disease is herein presented.

METHODS AND MATERIAL

Cardiac catheterization was carried out in the usual manner using light sedation (usually secobarbital, 0.1 Gm.). Pulmonary artery pressures were recorded using a Sanborn electromanometer and a direct writing four-channel Sanborn recorder. Peripheral arterial pressures were recorded via Statham strain gauge manometers. Simultaneous mixed venous (pulmonary artery) and systemic arterial (femoral or brachial) blood samples were analyzed for oxygen content by the Van Slyke method[24] and the spectrophotometric method of

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9 L G	C	44	14	26	100	30	50	20 2	15 6	77	12 0	3 6	289	8 0	5 3
	C_o	50	18	28				20 2	19 8	98	15 4	4 4	307	7 0	4 7
	O(p)	75	25	42	130	65	88	20 2	15 6	77	11 5	4 1	307	7 5	5 0
	O_o	62	20	34				20 2	19 9	99	15 9	4 0	289	7 2	4 8
10 L D (a)	C	48	27	37	130	80	100	19 1	15 4	83	10 4	5 5	253	4 6	2 7
	C_o	43	26					19 1	18 4	96	13 9	4 5		5 6	3 3
	O_o(c)	70	22	42				19 1							
L D (b)	CRPA	40	20	30											
	ORPA	70	35	50	150	90	120	20 2	16 7	83	11 4	5 3		4 0	2 7
	CLPA	40	20	30	150	90	120	20 2	18 2	90	11 7	6 5		3 2	2 1
	OLPA	60	30	42	160	100	125	20 2	17 4	86	11 7	5 7	215	3 8	2 5
	OLPA$_o$	50	25	35	175	100	125	20 2	20 0	99			210		
11 R H	C	45	25	33	100	75		20 1	16 4	82	13 6	2 8	360	12 8	7 2
	C_o	32	18	24				20 1	19 5	97	15 1	4 4		8 2	4 6
	O(p)	68	24	45				20 1	16 7	84	13 0	2 7	400	10 8	6 1
	O_o	69	22	42				20 1	19 4	97	15 3	4 1		9 8	5 5
	O_oE	78	28	56											
12 M H	C	54	31	40	150	77	105	17 6	16 0	91	11 2	4 8	188	3 9	2 5
	C_o	58	18	32	135	68	90	17 6	17 6	100	13 1	4 5	188	4 2	2 7
	O(p)	70	20	37	140	65	90	17 6	15 9	91	11 6	4 3	186	4 3	2 7
	O_o	64	15	35	135	68	90	17 6	17 6	100	13 0	4 6	186	4 1	2 6
13 V S	C	15	5	10								5 6	140		2 5
	O(c)	30	10	20								(Assumed)			(Assumed)
14 D D	C	24	9	15	105	75	88	16 1	14 3	89	9 4	4 9	239	4 9	3 2
	C_o	28	10	15				16 1							
	O_o(p)	44	20	30	105	75	88	16 1	15 8	98	11 2	4 6	271	5 9	3 8
	O_oE	60	20	35				16 1	16 1	100	10 0	6 1	453	7 4	4 8
15 W T	C	30	19	22	100	55	75	21 2	19 1	90	14 6	4 5	161	3 6	2 1
	C_o	30	15	20	105	70	80	21 2	21 0	99	15 6	5 4		3 0	1 7
	O_o(p)	58	36	38	125	75	100	21 2	19 0	90	13 6	5 4	214	4 0	2 3
	O_o	52	22	34	105	65	80	21 2	20 4	99	15 2	5 7		3 8	2 1
	O_oE	70	40	55	140	90	115	21 2	19 1	90	11 2	7 9	535	6 8	3 9
	CE	42	18												

C = control (unoccluded) O = unilateral pulmonary artery occlusion E = exercise, C_o on Collins spirometer, breathing 100 per cent oxygen O_o = occluded on oxygen O_oE = exercise while occluded on oxygen S = systolic D = diastolic M = means Cap = blood O capacity Art = arterial oxygen content % Sat = per cent saturation V = mixed venous (pulmonary artery) oxygen content AV = arteriovenous oxygen different O_2 = oxygen consumption in cubic centimeters per minute CO = cardiac output CI = cardiac index pressures are in mm Hg oxygen contents in volumes per cent

Where indicated following O (occlusion) (p) indicates occlusion of part of the pulmonary blood flow to the right lung either right lower lobe or right lower and middle lobes (c) indicates occlusion of the right main pulmonary artery In Case 10b first the right main pulmonary artery was occluded indicated by RPA and then the left indicated by LPA.

TABLE II. CATHETERIZATION DATA IN PATIENTS WITH CHRONIC PULMONARY DISEASE

		ARTERIAL PRESSURES						BLOOD OXYGEN CONTENT							
		PULMONARY			BRACHIAL										
CASE NO.		S	D	M	S	D	M	CAP.	ART.	% SAT.	V	A-V	O_2	CO	CI
1. J. B.	C	25	7	15	135	75	92	17.5	16.0	91	12.1	3.9	252	6.5	3.6
	C_o	25	7	15	140	80	98	17.5	17.3	99	13.0	4.3	252	5.9	3.2
	O_o(c)	32	10	20	140	85	100	17.5	17.7	100	13.2	4.5	252	5.6	3.1
	O_oE	41	15	27	145	100							468		
2. G. F.	C	20	5	10	100	65	80	19.8	18.2	92	14.0	4.2	434	10.3	5.6
	C_o	20	5	10				19.8	19.6	99	15.3	4.3	434	10.1	5.5
	O(c)	30	8	15				19.8	18.5	94	14.3	4.2	396	9.4	5.1
	O_o	25	5	13				19.8	19.4	98	13.7	4.7	396	8.4	4.5
3. V. M.	C	42	10	18				17.6	13.3	76	8.9	4.4	168	3.7	2.5
	C_o	38	12	22				17.6	17.2	97	12.7	4.5	168	3.7	2.5
	O(p)	61	18	32				17.6	14.7	84	9.2	5.5	174	3.2	2.2
	O_o	56	10	26				17.6	17.3	98	12.9	4.4	174	4.0	2.7
4. G. H.	C	30	18	25	96	60	72	22.4	19.6	88	13.3	6.3	361	5.7	4.0
	C_o	42	12	22				22.4	22.4	100	16.4	6.0	361	6.0	4.2
	O(c)	59	35	40	130	95		22.4	18.7	84	11.7	7.0		5.2	3.6
5. M. H.	C	48	18	33				17.1	13.4	79	7.9	5.5	460	8.4	4.4
	C_o	43	18	30				17.1	15.0	88	9.6	5.4	460	8.5	4.4
	O_o(c)	62	25	40				17.1	13.7	80	6.7	7.0	716	10.2	5.4
6. H. W.	C	55	5	25				19.8	15.5	78	12.8	2.7	400	14.8	8.6
	C_o	42	13	22				19.8	19.5	98	14.5	5.0	400	8.0	4.6
	O(p)	65	16	40				19.8	19.1	96	14.7	4.4	470	10.7	6.2
7. B. S. (a)	C	65	30	45	125	60	80	18.2	12.2	67	5.3	6.9	170	2.5	1.4
	C_o	65	30	45	125	75	82	18.2	17.1	94	11.8	5.3		3.2	1.9
	O(c)	66	30	45	125	75	82	18.2	13.2	73	6.3	6.9		2.5	1.4
	O_o							18.2	17.7	98					
B. S. (b)	C	54	30	40	100	70	78	18.4	14.8	81	9.6	5.2	150	2.9	1.7
	CE	60	30	44				18.4	15.3	83	8.8	6.5	200	3.1	1.8
	O(c)	58	28	40	125	75	80	18.4	14.9	81	9.2	5.7	150	2.6	1.5
	O_o	54	30	40				18.4	17.6	96	12.0	5.6	150	2.7	1.6
8. M. H.	C	82	28	40				19.8	15.5	78	11.7	3.8	300	7.9	3.9
	C_o	72	22	38				19.8	19.5	98			(Assumed)		
	O(p)	92	30	46				19.8	15.2	77	12.1	3.1	300	9.7	4.8
	O_o	84	20	42				19.8	19.3	97	14.0	5.3	300	5.7	2.8

23. E. W.	C	38	20	28	100	50	60	16.6	13.9	84	8.0	5.9	196	3.3	2.1
	C_o	34	18	27				16.6	14.8	89	9.0	5.8		3.4	2.2
	O(c)	45	18	30				16.6	14.5	88	8.9	5.6	250	4.5	2.9
	O_o	58	27	35				16.6	15.4	93	10.5	4.9		5.1	3.2
	O_oE	62	40	50				16.6							
24. O. B.	C	38	16	28	100	25	50	15.4	13.7	89	9.9	3.8	254	6.7	4.4
	O(c)	72	20	38				15.4	14.0	91	9.3	4.7	254	5.4	3.4
	O_o	70	18	35	130	30	65	15.4	14.8	96	12.2	2.6	254	9.8	6.2
25. E. O.	C	65	32	45				17.6	16.0	91	11.1	4.8	234	5.6	3.6
	C_o	63	30	44				17.6	16.5	91	9.1	7.4	234	3.2	2.1
	O(c)	65	30	46				17.6	15.9	90	10.0	5.9	378	6.4	4.2
	O_o	63	30	44				17.6	16.8	96	10.2	6.6	378	5.7	4.1
26. R. H.	C	60	30	40				16.4	14.1	86	7.9	6.2	226	3.6	2.1
	C_o	60	30	40				16.4	15.9	97	9.8	6.1	226	3.6	2.1
	CE	85	50	60				16.4							
	O(c)	70	35	50											
27. W. B. (a)	C	70	32	50	120	75	90	20.6	17.2	84	13.5	3.7	320	8.7	4.0
	C_o	62	32	47	115	70	85	20.6	19.0	93	15.6	3.4		9.4	4.2
	O(p)	60	29	42	110	65	85	20.6	18.1	88	13.6	4.4		7.3	3.3
	O_o	62	25	42			88	20.6	19.6	96	15.7	3.9		8.2	3.8
	O_oE	80	50	68									532		
W. B. (b)	C	42	25	32	100	50	75	20.1	17.1	85	13.7	3.4	345	10.1	4.7
	C_o							20.1	18.8	94	14.3	4.5		7.7	3.6
	O(p)	45	30	35				20.1	17.3	86	13.1	4.2	380	9.0	4.2
	O_o	42	24	30				20.1	19.0	95	14.1	4.9		7.9	3.6
28. D. E.	C	82	30	52	125	50	75	18.6	16.9	91	8.0	8.9	471	5.3	2.7
	C_o	80	25	51	125	60	78	18.6	18.3	99	10.8	7.5	471	6.3	3.2
	O(p)	82	40	55	120	60	75	18.6	17.0	92	9.4	7.6	525	7.0	3.6
	O_o	70	30	45	120	60	75	18.6	18.4	99	10.2	8.2	525	6.5	3.3

TABLE II—CONT'D

CASE NO.		ARTERIAL PRESSURES						BLOOD OXYGEN CONTENT							
		PULMONARY			BRACHIAL										
		S	D	M	S	D	M	CAP.	ART.	% SAT.	V	A-V	O_2	CO	CI
16. W.B.	C	56	20	30	110	40	65	17.6	14.9	85	10.0	4.9	365	7.5	4.2
	C_o	50	16	28				17.6	16.8	96	11.8	5.0	365	7.3	4.1
	O(c)	85	22	58	130	50	75	17.6	14.0	80	8.2	5.8	544	9.4	5.3
	O_o	80	20	56				17.6	16.9	96	11.4	5.5	544	9.9	5.5
	OE	90	20	46				17.6	13.0	74					
17. C.E.	C	39	20	30	150	90	110	20.2	18.5	92	14.7	3.8	370	9.8	4.9
	C_o	35	15	26	150	90	110	20.2	20.2	100	13.2	7.0	370	5.3	2.7
	O(c)	38	18	28	150	90	110	20.2	18.1	90	16.0	2.1	370	18.0	9.2
	O_o	40	15	28	150	90	110	20.2	20.2	100	17.0	3.2	370	11.6	5.8
	O_oE	60	25	38	150	90	110	20.2	20.2	100	13.8	6.4	1060	16.6	8.3
18. B.L.	C	55	14	28	100	40	68	15.7	14.4	92	8.1	6.3	308	4.9	3.1
	C_o	53	18	30	90	35	62	15.7	15.2	97	8.8	6.4	308	4.8	3.0
	O(c)	85	27	43	90	40	63	15.7	9.6	61					
	O(p)	80	35	42				15.7	11.1	71	6.9	4.2	346	8.3	5.3
	$O_o(p)$	75	25	39	120	50		15.7	14.6	93	8.6	6.0	346	5.8	3.7
19. F.B.	C	25	8	12				17.3	16.0	93	12.0	4.0	281	7.0	4.0
	C_o							17.3	16.9	99					
	O(c)	34	8	15											
20. F.M.	C	28	10	17	100	55		12.5	10.8	86	6.2	4.6	240	5.2	3.8
	$O_o(p)$	35	18	25				12.5	11.8	94	7.5	4.3		5.6	4.0
	O_oE	60	25					12.5	11.9	95	5.5	6.4	530	8.3	5.9
21. G.W.	C	32	20	26	110	75	90	13.6	11.1	82	7.1	4.0	258	6.5	4.2
	C_o	26	12	20	120	75	95	13.6	12.7	93	8.7	4.0			
	O(c)	32	20	26	115	80	95	13.6	11.7	86	7.6	4.1		6.3	4.1
	O_o	30	18	24	130	80		13.6							
22. F.B.	C	35	12	20	100	60	75	13.0	11.1	85	6.0	5.1	313	6.1	3.7
	C_o	35	15	20	105	65	75	13.0	12.6	97	7.9	4.7	221	4.7	2.9
	O(c)	40	15	23	100	60	75	13.0	11.2	86	6.4	4.8	258	5.4	3.3
	O_o	35	10	20	110	60	80	13.0	12.8	98	8.1	4.7		5.5	3.4
	O_oE	40	10	24	125	80	80	13.0	12.7	98	6.6	6.1	442	7.3	4.5

artery resulted in dyspnea, increased cardiac index, rise in pulmonary artery pressure, and fall in peripheral arterial oxygen saturation. The significance of this response is presented in the discussion.

On the other hand, in other patients (Cases 7 and 17) unilateral occlusion was tolerated much better than expected. Except in those instances noted, there were no subjective symptoms associated with occlusion.

Intracardiac Pressures.—In Fig. 1, the flow-pressure relationships are plotted for the *emphysema group* (chronic diffuse and bullous or secondary). In this group there is no correlation between the control main pulmonary artery pressure and the pressure following occlusion. The increase in flow through the unoccluded lung was attended by a significant increase in main pulmonary artery pressure in some patients (Cases 3, 4, 9, and 10b). However, in others (1, 2, 7a, and 7b), there was little or no increase. In 2 patients (Cases 10a, 12) the pressure actually fell during occlusion.

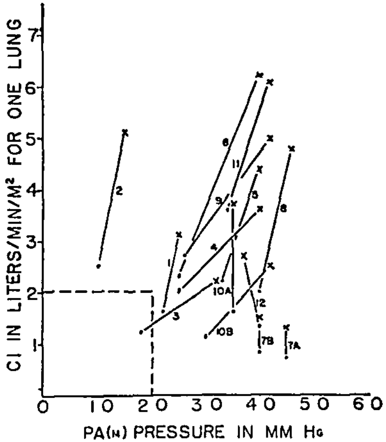

Fig. 1.—Flow-pressure relationships in patients with emphysema. Pulmonary artery mean pressure plotted against cardiac index for one lung. Symbols: ● = subject at rest; x = during unilateral pulmonary artery occlusion. Upper limits of normal at rest indicated by dotted lines.

In Fig. 2 are plotted the flow-pressure relationships existing in those cases of *granulomatous disease.* In every case, increase in flow by contralateral occlusion produced a marked increase in pulmonary artery mean pressure. Still further increases were produced with exercise. It should be pointed out that the increase in pressure was predominantly systolic with less change in the diastolic phase.

In Fig. 3 a similar plot is presented for those cases of combined *left ventricular heart disease and pulmonary disease.* In all but 1 case there was no pressure change with unilateral increase in flow. In the instance where pressure did rise significantly, granulomatous disease of the lung was also present.

output was calculated utilizing the Fick principle. Control values were obtained before occlusion of the pulmonary artery by the inflatable cuff, and repeated following this maneuver. Control and occluded values were also determined with the patient breathing 100 per cent oxygen. In many patients, mild exercise was carried out and the effects noted. No attempt was made to attain absolute basal conditions during these previous studies, although a "steady study" was achieved.

The technique used in our laboratory has been presented.[2] In the series herein reported, all the patients had pulmonary disease with varying degrees of functional impairment, ranging from asymptomatic to completely incapacitating. In Table I are listed the subjects studied, with diagnosis and some of the standard pulmonary ventilatory studies. They are grouped according to diagnosis, as follows: (1) chronic diffuse, emphysema, with or without cor pulmonale; (2) bullous emphysema (complicated) or emphysema secondary to other pathologic processes; (3) granulomatous lung disease of various etiologies; (4) primary bronchiogenic carcinoma; and (5) combinations of cardiac disease involving the left ventricle and/or the mitral valve and pulmonary disease. Control catheterization findings were consistent with the grouping in that all of the patients with pulmonary disease without significant left ventricular or mitral valve disease had normal wedge (pulmonary capillary) or distal (lacunar) pressures. The latter pressure is that recorded beyond the inflated balloon. In all our studies, wedge and distal pressures have been almost identical. Patients with combined disease all had significantly elevated wedge or distal pressures. The subject of distal pressures and their significance will be discussed in a separate publication.

In most patients, complete unilateral occlusion was achieved as described. In some instances, however, as indicated in Table II, only partial occlusion of one lung was obtained, fortuitously or by intent. Usually, when the entire right or left lung was not occluded, this was due to the fact that the balloon was carried out beyond the desired point in the main subdivision of the pulmonary artery. In certain cases, because of severity of symptoms or initial marked elevation of pulmonary artery pressure, it was considered inadvisable to occlude the main branch.

RESULTS

In Table II are listed the pertinent findings in the cases studied. Unilateral occlusion was well tolerated in all but 3 patients (Cases 4, 16, and 18). Obvious difficulty had been anticipated in these 3. Case 4 was an individual with far-advanced emphysema who had become dyspneic even at rest. Case 16 had granulomatous lung disease of undetermined etiology (proved by lung biopsy), with dyspnea on slight exertion and marked impairment of ventilatory function. Case 18 was an elderly man with repeated episodes of chest pain, cyanosis, and shortness of breath. Chest x-ray studies suggested emphysema but pulmonary function studies were not consistent with this diagnosis. It was felt that he had developed the syndrome of alveolar-capillary block due to repeated pulmonary embolization.[9] In all 3, occlusion of the right main pulmonary

The patients with carcinoma responded in a normal manner with a slight rise in main pulmonary artery pressure following occlusion. These pressures shown in Table II were within normal limits.

Patients 7, 10, and 27 were studied before and after definitive therapy. The first of these, before treatment for severe emphysema and cor pulmonale, had an end diastolic pressure in the right ventricle of 20 mm. Hg which decreased to 10 mm. Hg following therapy. Case 10 was studied before and after surgery for removal of bullae and Case 27 before and after mitral commissurotomy. It is notable that in the latter case, following commissurotomy, both wedge and pulmonary artery pressure had fallen and the response to occlusion was significantly altered.

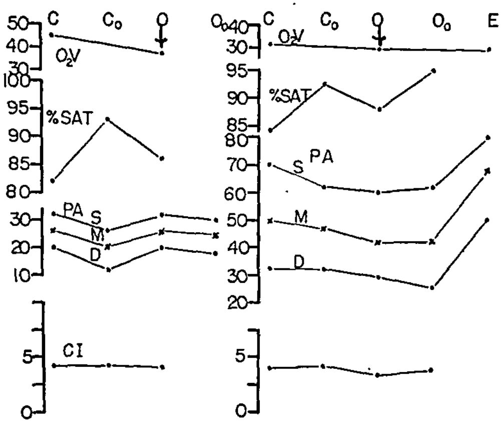

Fig. 4.—Effect of uni[illegible] n in 2 patients showing rise in arterial saturation during occlusion. Symbols: [illegible] C_o = control on 100 per cent oxygen; O = unilateral occlusion; O_o = 100 per cen[illegible]hing during occlusion; E = exercise; O_2V = ratio of oxygen consumption to min[illegible] in cubic centimeter per liter; % Sat. = systemic arterial saturation; PA = [illegible]ry pressure in mm. Hg; S = systolic; M = mean; D = diastolic; CI = cardia[illegible]s per minute per meter squared.

In Case 21 (*left*) the [illegible] are normal for the O_2V and CI with slight elevation of the PA pressure and ma[illegible] in arterial O_2 saturation. On 100 per cent oxygen, the expected fall in PA p[illegible]crease in O_2 saturation occurred. With occlusion, however, the O_2 saturation [illegible]ared to the control value without other significant change. In Case 27 (*right*[illegible] are normal for the O_2V but a markedly elevated PA pressure was present to[illegible]omewhat increased CI and again a marked diminution in arterial O_2 saturati[illegible]er cent oxygen, there was a fall in PA pressure and increase in O_2 saturatio[illegible]h occlusion, the O_2 saturation rose significantly concomitant with a fall in PA [illegible]CI toward normal as compared with control values. One hundred per cent oxy[illegible] further increase in arterial O_2 saturation without other significant change an[illegible] noted to cause a marked increase in PA pressure.

Peripheral Arterial Pressures.—No significant change was observed following occlusion.

Peripheral Arterial Oxygen Saturation.—In most cases, no change occurred. In at least 2 patients (21 and 27), peripheral arterial saturation increased following occlusion, as demonstrated by the hemodynamic response in Fig. 4. In both cases, a significant rise can be observed and, in Case 27, this was associated with a fall in cardiac index as well as pulmonary artery pressure. Both had disease involving predominantly the occluded side.

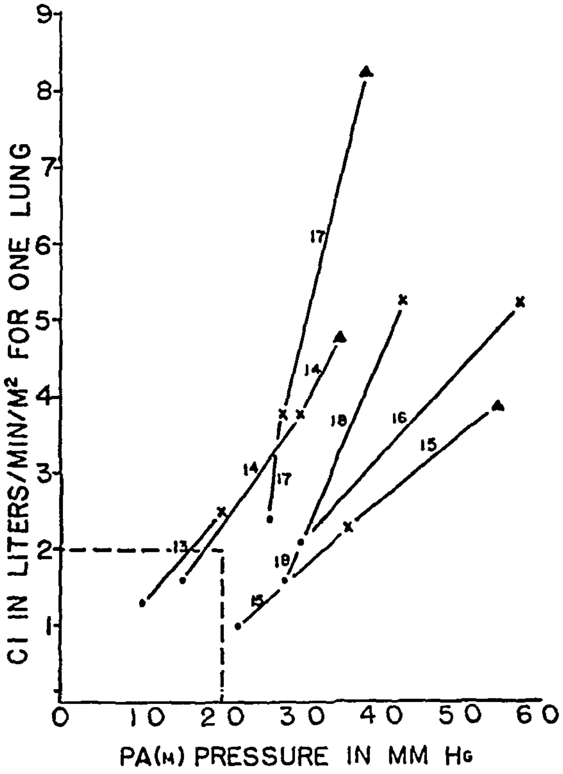

Fig. 2.—Flow-pressure relationships in patient with granulomatous disease. Symbols as before; ▲ = exercise during unilateral occlusion.

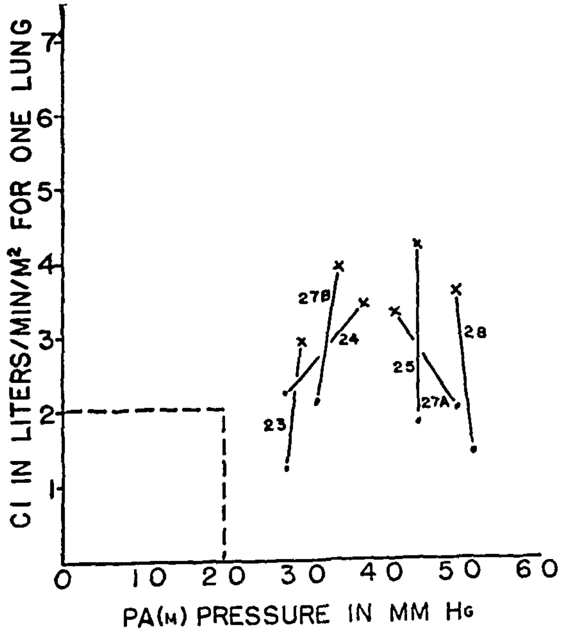

Fig. 3.—Flow-pressure relationships in patients with combined cardiac and pulmonary disease. Symbols as before.

by the disease process. In the patients with myocardial failure, however, despite an apparently increased resistance, this resistance was not due to pulmonary vascular change since increase in flow was not accompanied by increase in pressure. Thus, pulmonary resistance in these cases was greatly diminished following occlusion. In the emphysema group, many patients showed fixed resistance presumably on the basis of anatomic changes. In others, resistance was greatly diminished following occlusion. This suggests that structural changes in the pulmonary vessels need not have occurred despite initial high resistance.

DISCUSSION

The study of pulmonary hemodynamics in patients with chronic pulmonary disease yields widely varying results. The control pressure in the pulmonary artery may vary from normal to three times normal. It cannot be inferred, however, that significant pulmonary arteriolar disease exists solely on the basis of an elevated resting pressure. Nor can it be assumed that a normal or only slightly elevated pressure precludes the existence of disease. As has been demonstrated, sudden deletion of approximately 50 per cent of the pulmonary vascular bed, with resultant doubling of flow through the remaining portion, need not result in any significant rise in pressure whether the resting pressure is normal or even greatly elevated. Cournand[11-13] and Hickam and Cargill[14] have reported similar control elevation of pressures in patients with chronic pulmonary emphysema. Riley and co-workers,[15] by means of exercise, have demonstrated marked increase in pulmonary artery pressures in such cases. However, certain important differences exist between exercise as a test and unilateral occlusion. As pointed out earlier, the method described in effect increases flow (unilaterally) without over-all increase in cardiac output while, with exercise, over-all cardiac output is increased. Furthermore, factors involved in exercise such as increases in intrapleural pressures, changes in position, increased heart rate, reflex changes, metabolic changes, etc., differ considerably from those described.

Since pulmonary hypertension in emphysema is not necessarily due to arteriolar change per se, the cause of the hypertension must be re-evaluated. Increased intra-alveolar or intrapulmonic pressure in such patients presumably accounts to a considerable degree for the elevation of the pulmonary arterial pressure. Positive intrapleural pressures have been demonstrated to reach a level of 20 mm. Hg or more in emphysema.[16] These factors could increase resistance to pulmonary blood flow necessitating a rise in pressure to maintain circulation throughout all phases of the respiration and inherent vascular change need not be present. Perhaps the best explanation is that of the "critical closing pressure" discussed by Burton.[17]

As suggested by Lillienthal and Riley,[18] the concept of the "critical closing pressure" can be applied to pulmonary hemodynamics. According to this concept, in order to maintain blood flow through a vessel, the intraluminal pressure [illegible] his must be greater than those factors which would tend to [illegible] the vessel wall [illegible] ion Such factors include the elastic and muscular tensio [illegible] as well as extravascular factors. It is presumed t [illegible] disease states, such as emphysema, there may be a [illegible]

In Cases 4, 16, and 18, previously described, the opposite response occurred, namely, a fall in peripheral arterial oxygen saturation associated with a rise in cardiac index and pulmonary artery pressures. Fig. 5 demonstrates this response. All patients had severe chronic diffuse pulmonary disease and experienced dyspnea on occlusion.

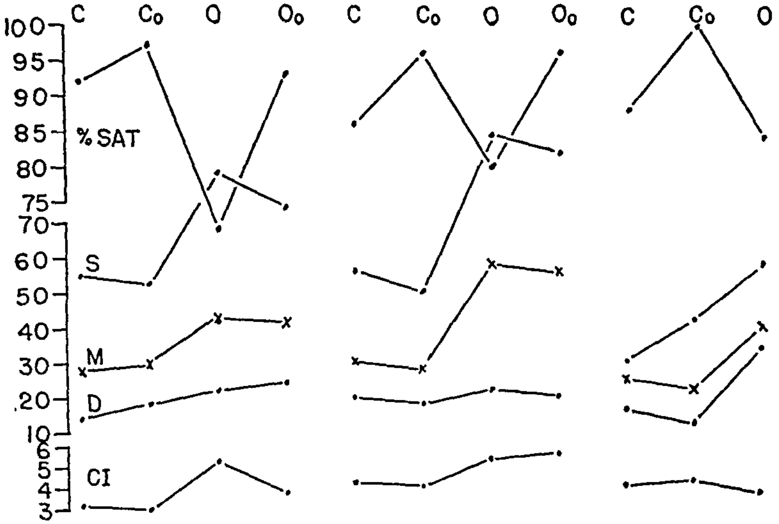

Fig. 5.—Effect of unilateral occlusion in 3 patients showing a decrease in arterial O_2 saturation. In Case 18 (*left*) the control O_2 saturation was slightly diminished, the PA pressure elevated, and the CI normal. On 100 per cent oxygen the arterial saturation rose to normal. With occlusion, the arterial O_2 saturation fell markedly associated with a sharp rise in PA pressure and CI. On 100 per cent oxygen, the saturation rose to control levels and the PA pressure and CI diminished.

Case 16 (*middle*) showed an initial decrease in O_2 saturation, elevated PA pressure and CI. On 100 per cent oxygen, the arterial saturation rose to normal levels. With occlusion the arterial O_2 saturation fell with a sharp increase in PA pressure and CI. One hundred per cent oxygen increased the saturation to normal once again but effected little change in pressure or CI.

In Case 4 (*right*), the depressed control O_2 saturation fell with occlusion again associated with a marked increase in PA pressure.

Cardiac Index.—This did not change significantly except in those cases described. Generally, for the purposes of this presentation, it can be assumed that there is approximately equal distribution of pulmonary blood flow to both lungs. However, in the presence of unilateral disease with atelectasis, a significant degree of shunting away from diseased lung may occur naturally.[10] In such cases, in which blood flow has already been redistributed, unilateral occlusion (of the diseased side) may only result in a relatively slight further redistribution.

Oxygen Consumption.—No significant change occurred except in those cases where dyspnea and resultant anxiety caused an increase.

Minute Volume.—Again there was no significant change although in many cases there was a slight rise. This is of particular importance since the occluded lung continues to ventilate but does not exchange gases.

O_2V.—Since the minute volume and over-all oxygen consumption did not change significantly and all gaseous diffusion was occurring in the unoccluded lung, the O_2V for that lung was approximately doubled.

Resistance.—As indicated in the figures showing the flow-pressure relationships, resistance was fixed in those patients with granulomatous lesions, presumably as a result of anatomic involvement of the small pulmonary vessels oxygen saturation may occur because of venous admixture in the lung. This situation exists when poorly ventilated alveoli are receiving a disproportionate perfusion. The result is failure of adequate oxygenation of blood from such areas and the equivalent of a right-to-left shunt. Methods for assessing the extent of such effects are indirect and often it is impossible to distinguish between faulty ventilation-perfusion relationships and disease of the alveolar capillary membrane with impaired diffusibility.

Utilizing the method described, it seems likely that localized areas of underventilation might be temporarily deprived of their pulmonary arterial supply. If the remaining unoccluded lung were normal, this maneuver should produce a rise in arterial oxygen saturation since all pulmonary capillary blood would be exposed to normal alveolar oxygen tensions. This procedure would have the added advantage of localizing such defective areas anatomically.

This effect is demonstrated in 2 cases with unilateral pulmonary disease and in 2 cases of diffuse pulmonary disease (Cases 3, 7, 21, 27). In these instances, occlusion of the pulmonary artery resulted in significant increases in arterial oxygen saturation. Paralleling this increase in saturation, there was observed a fall in pulmonary artery pressure and cardiac output. It seems apparent that areas of venous admixture were obliterated and over-all function improved.

In patients with bilateral severe chronic pulmonary disease, the opposite situation is also possible. Where arterial desaturation exists because of diminished ventilation-perfusion ratios, occlusion of one lung may accentuate the venous admixture occurring in the other. This situation obtains in 2 cases (Cases 8 and 9), where instead of a rise in arterial O_2 saturation there was a further fall. This response must be differentiated from that due to exceeding the maximal diffusing capacity of the unoccluded lung which will be discussed subsequently. In both instances, the further decrease in arterial O_2 saturation was associated with an increase in the pulmonary artery pressure, increased cardiac output and hyperventilation, necessitating deflation of the cuff in one case. On exposure to 100 per cent oxygen, both patients showed the expected rise to normal arterial O_2 saturation.

Diffusion.—Determinations of diffusing capacity of the lung at rest have shown wide variation and changes in diffusion have been demonstrated with exercise.[22] The maximal diffusing capacity of the lungs seems more promising as a basis for comparison in normal subjects and in those with pulmonary disease.[6, 23] Riley and co-workers have described such a method for maximal O_2 diffusing capacity, utilizing hypoxia and exercise. The factors limiting such determinations particularly in the presence of pulmonary disease may present considerable difficulty. Defects such as poor intrapulmonary mixing of gases, uneven ventilation-perfusion relationships, and circulatory failure may each cause a reduction in maximal diffusing capacity.

In this respect, of the cases studied, Case 7 is of particular interest. This patient initially presented a relatively normal resting arterial O_2 saturation and a normal cardiac index. Pulmonary artery pressure was elevated. Ventilation studies did not seem to indicate that obstructive pulmonary [illegible]ase was the predominant difficulty. When occlusion of the ri[illegible] p [illegible] critical closing pressure because of the extravascular factors mentioned. Thus, the presence of pulmonary hypertension at rest does not necessarily imply anatomic pulmonary vascular change. Furthermore, a slight rise in pressure may open accessory channels allowing increases in flow out of proportion to the pressure increment.

The pressure response in patients with granulomatous disease was more predictable and confirms the findings of others.[4, 13, 19] In these cases, whether the resting pressure was normal or greatly elevated, occlusion, with unilaterally increased flow, produced a significant increase in pressure indicating considerable fibrosis of the pulmonary arterial tree, loss of elasticity, and hence a fixed resistance. The tremendous expansibility of the normal pulmonary vascular bed has been emphasized by Cournand[12] and in a previous publication.[2] Limitation of this distensibility by fibrotic pulmonary lesions can be fairly well defined by the method described.

It should be emphasized that the level of the resting pulmonary artery pressure does not necessarily reflect the status and distensibility of the pulmonary vessels. An elevated pressure is not necessarily a contraindication to resectional lung surgery. On the other hand, patients with normal pressures at rest may have no reserve with respect to accommodation to increased blood flow and are, therefore, poor risks for such surgery. Deletion of any segment of their pulmonary vascular bed would result in a significant increase in pressure.

In patients with combined pulmonary disease and cardiac disease involving the left ventricle or mitral valve, the evaluation of relative causes of disability may be extremely difficult. In the group studied, all had elevation of both pulmonary capillary (or wedge) pressure and pulmonary artery pressure. Only one, however, showed any significant rise with occlusion. Exercise in such cases increases the cardiac output. As a result, pulmonary artery pressure rises but this response does not aid in determining whether the increase in pressure is due to resistance in the left atrium or in the pulmonary vasculature. By means of occlusion, flow can be increased through a part of the lung without increase in over-all cardiac output thus facilitating the study of these two factors independently. It may be concluded from the cases shown that elevation in pressure was predominantly, if not entirely, due to increased left atrial pressure since no significant elevation in pulmonary artery pressure occurred with increase in flow. In these cases, the pulmonary vasculature is normal and can accommodate an increase in flow with a normal slight increase in pressure. In one case, however, the marked increase in pressure was evidence for the existence of increased resistance in the pulmonary vascular bed. Granulomatous pulmonary disease was subsequently proved by biopsy.

The importance of this relationship in patients with congenital and acquired heart disease, particularly in determining if secondary pulmonary arteriolar hypertrophy exists in patients with mitral stenosis, will be presented in a separate report.

Uneven Ventilation—Blood Flow Relationship.—The problems relating to ventilation-perfusion relationships in the lung have received much attention recently.[3, 5, 20, 21] It is now well appreciated that significant reduction in arterial

We wish to thank Dr S Wolpaw and Dr R I Newman for their assistance in studying the patients discussed, and Mrs M Rizika and Mrs D Fader for their technical assistance

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artery was carried out, there was a marked fall in arterial O_2 saturation and an increase in pulmonary artery pressure and cardiac output. The O_2 consumption, however, did not rise despite marked dyspnea. On 100 per cent oxygen, arterial saturation rose to normal levels and cardiac output fell to normal. Although the factor of uneven ventilation blood flow has not been entirely excluded, it is felt in this instance that diversion of the extra blood flow through the unoccluded lung exceeded the maximal diffusibility of that lung due to specific disease of the alveolar capillary membrane. The increase in cardiac index and hyperventilation are explainable on the basis of hypoxia but, at the same time, indicate that neither cardiac output nor alveolar ventilation was a limiting factor in this patient's oxygen diffusibility. The normal resting arterial O_2 saturation rules out significant venous admixture. The maximal diffusing capacity in this patient therefore appears severely limited and can be exceeded merely by reducing the area of pulmonary membrane available for diffusion.

The application of this technique in determining maximum diffusing capacity of a single lung is obvious, particularly with respect to delineating the limiting factors in such determinations.

SUMMARY

1. Twenty-eight patients with chronic pulmonary disease have been studied by the method of temporary unilateral pulmonary artery occlusion using a special triple-lumen catheter with an inflatable cuff.

2. It is shown that the resting or control pulmonary artery pressure is not necessarily an index of the pulmonary circulatory reserve (expansibility). Without increasing cardiac output, approximate doubling of unilateral pulmonary blood flow may or may not be attended by significant increase in pressure, irrespective of the level of control pulmonary artery pressures.

3. It is suggested that in many cases of chronic pulmonary disease (without cardiac disease involving the left ventricle or mitral valve) pulmonary hypertension must be due to causes other than organic arteriolar disease, such as increased intrapleural and intrapulmonic pressures. The concept of a "critical closing pressure" is discussed in this regard.

4. In cases of mitral stenosis or left ventricular failure in combination with chronic pulmonary disease, this method has proved valuable in the study of pulmonary hemodynamics with particular reference to the status of the pulmonary arterioles.

5. The problem of uneven ventilation/blood flow relationships, where significant venous admixture exists, has been presented. It has been demonstrated that such areas can be obliterated by occlusion of the pulmonary artery to that segment. Resultant decrease in venous admixture has been demonstrated by a rise in arterial O_2 saturation.

6. Possible applications of temporary pulmonary artery occlusion in the determination of maximal O_2 diffusing capacity are discussed and particularly with reference to possible delineation of factors involved in such determinations.

A number of reports in the foreign literature give evidence that collapse methods, such as pneumothorax and thoracoplasty, when aided by an antituberculosis drug regimen give immediate and short-term results comparable to the excellent results achieved by excisional surgery.

The early and late results of 90 thoracoplasties performed on the ward service at Montefiore Hospital, in New York City, from 1935 to 1940, have already been reported.[1, 2] To these have been added the data of an additional consecutive 90 patients from the same service operated upon by the authors from 1940 to 1947. (It was entirely accidental that there were an equal number of patients in each series.) The total number of patients treated by extrapleural thoracoplasty, primarily for pulmonary tuberculosis, is 180. Except for a few cases in the first series, those patients in whom an empyema overshadowed the pulmonary disease were excluded from this report.

The extent of the follow-up was such that it took a number of years to complete. We have a 100 per cent follow-up, i.e., the status of every patient has been ascertained. Four patients who had been operated upon before 1941 were last contacted in 1953. The status of the remaining number was determined in 1954, 1955, or 1956, the date of preparation of this paper. Most of the follow-up data were obtained from the Altro Work Shops, Inc., from the Montefiore Hospital Follow-Up Clinic, from Board of Health Clinics, and various other clinics in New York City, as well as those in other cities and states, and from private physicians. In only a small number of cases were the reports obtained directly from the patients. These were patients operated upon many years ago, who were no longer under routine medical care. The reports on sputum examinations were based on cultures; a few were made on concentrates.

We have been more concerned with those patients in whom thoracoplasty was unsuccessful rather than with those who were cured. We have analyzed the case histories with a view to determining the reason for failure to cure and how this cause for failure could be avoided today. When these patients were operated upon, there was no alternative good procedure which could be used. Penicillin became available to only a few patients in this series; none had the advantage of the antituberculosis drugs; and many of the aids to surgery and to anesthesia which are used routinely today were not yet available.

There were 9 postoperative deaths; 6 of these occurred in the first series of 90 patients and 3 in the last 90. Of the first 6 deaths, 3 were due directly, or indirectly, to a pleural tear occurring during operation. These tears were packed with gauze, and the complications resulted eventually in a fatality. The fourth death was caused by a phlegmon of the back. The fifth patient died thirty minutes after operation and, at autopsy, a contralateral atelectasis was found. The sixth death was that of a patient who died twelve hours postoperatively with convulsions, tachycardia, hypertension, and delirium. At autopsy, an unsuspected pheochromocytoma of the adrenal was found. Of the 3 deaths in the second group of 90 cases, one was due to operative shock and occurred twelve hours after a first-stage procedure; in the other 2, the causes of death were unexplained. One, a 46-year-old man, was found dead on the third postoperative day after a two-day uneventful course; the

EXTRAPLEURAL THORACOPLASTY IN THE TREATMENT OF PULMONARY TUBERCULOSIS

An Eight- to Twenty-One-Year Follow-up Study

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UNTIL the discovery of streptomycin, bed rest and collapse therapy were the mainstays in the treatment of pulmonary tuberculosis. Of the various forms of collapse therapy, artificial pneumothorax and extrapleural thoracoplasty were the procedures upon which we depended for a number of decades. During that period many thousands of extrapleural thoracoplasties of diverse types were performed. As the adjuvants to surgery developed and surgical technique improved, it was possible to obtain increased pulmonary collapse, and there was a decrease in surgical complications; consequently, operative results were decidedly better.

With the introduction of the various antituberculosis drugs, a sudden change in surgical therapy developed. Collapse therapy quickly yielded to drug therapy. When a cure was not obtained by the use of drugs alone, then pulmonary resection became the procedure of choice. It was the abruptness of this change which, in a great measure, robbed collapse procedures, especially thoracoplasty, of the opportunity to demonstrate the good results they might achieve when used in conjunction with the newly found drugs. So few collapse procedures are being performed in this country today, that it will be many years before any one center will accumulate sufficient data to compare the results of the various collapse methods with those obtained by excisional therapy, when both have been performed with the aid of the new therapeutic agents.

Because of these circumstances, we feel that it is of the utmost importance to evaluate precisely the long-term results of collapse methods and especially extrapleural thoracoplasty. The known chronicity of pulmonary tuberculosis and its proclivity for reactivation many years after apparent cure make only long follow-up periods of value. With this knowledge in hand, we should be able to postulate to some degree what our long-term results would be were thoracoplasty performed with the additional benefits that could be derived from the simultaneous use of our antituberculosis drugs. Only in this way will we be able to compare our long-term results of excisional therapy (when we have them) with what could be expected from collapse therapy if performed under a similar drug therapy regimen.

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this They are, however, considered as poor results of thoracoplasty because of positive sputum. It is most unusual today to have to operate upon a patient in poor condition. Practically all of the patients who have pulmonary tuberculosis respond, at least to some degree, to the antituberculosis drugs; the tempo of the disease is thus lessened and the patient can be the better prepared for surgery.

There were also 6 patients in whom the poor result of thoracoplasty could be ascribed to incomplete surgery. In 4 of these patients, a deterioration in the general condition prevented the second-stage operation; one patient committed suicide; and the sixth patient refused further surgery, became psychotic, and was discharged for disciplinary reasons.

Today, with the use of the antituberculosis drugs, endobronchial disease and its complications are well controlled and are not a problem in surgery. Not all the patients in our series were bronchoscoped, therefore, many cases of endobronchial disease were not diagnosed. Four patients, known to have endobronchial disease, had unsuccessful thoracoplasties.

The spread of pulmonary tuberculosis to other segments of the same lung or to the other lung was one of the hazards of thoracoplasty. Such a spread usually delayed the second-stage procedure for so long a period that it rarely was effectual. In some instances, the postoperative spread was the beginning of the ultimate fatal progressive tuberculosis Today, under drug therapy and with improved surgical technique and intratracheal anesthesia, postoperative spreads are rare indeed.

Each of the remaining classifications listed under the reasons for the unsuccessful thoracoplasties embraces too few cases to justify discussion. It is apparent, however, that today these patients would not be treated by collapse therapy, and the use of the antituberculosis drugs would in all probability eradicate the reason for failure.

In only 3 of the 38 cases of unsuccessful thoracoplasty were we unable to find some apparent reason for the failure of operation. With this past experience to guide us and with the use of the antituberculosis drugs and our improved techniques in surgery and anesthesia, if thoracoplasty were performed today upon a well-chosen series of patients, a very high percentage of permanent cures would be achieved.

The following objections to thoracoplasty have been raised: (1) the operation causes a deformity; (2) some patients have persistent cough and expectoration due to the fibrosis and bronchiectasis which are the end results of pre- and postoperative healing; (3) the diseased pulmonary tissue is still present in the body and may not undergo complete healing, thereby becoming a source for later infection; and (4) there is a reduction in pulmonary reserve and the patient later succumbs to cor pulmonale.

In our follow-up study, the question of deformity and arm and shoulder motion was thoroughly investigated. Scoliosis, as seen on the roentgenogram, may appear to be much more marked than it is clinically Many of our patients had a mild degree of scoliosis, but in none was it incapacitating and as far as we could determine, it was never a cause of mental disturbance to be independent of the disease which, originally present on the operated side, has remained arrested. Subsequent careful study of the preoperative roentgenograms frequently reveals that contralateral disease was present at the time of thoracoplasty (twenty of the unsuccessful thoracoplasties). Theoretically, such a reactivation should not mean that the operation was not a success, but we have classified these patients as having had unsuccessful thoracoplasties, in which group they are listed.

In our series, operation was not performed unless the contralateral disease was presumed to be stable and inactive. Most of these patients did not have the benefit of tomographic examination, therefore, the status of the contralateral pulmonary disease could not be established as accurately as it is today. Now, with the use of the antituberculosis drugs, contralateral disease can be better controlled prior to, and after, surgery and the likelihood of late reactivation will be lessened.

It was common knowledge that giant cavities and tension cavities were not readily closed by thoracoplasty. Yet, having nothing better to offer (we did not find the Monaldi suction drainage procedure of much value), many of them were treated by thoracoplasty. In a very small number was closure obtained. These cavities, both giant and tension, usually secondary to endobronchial tuberculosis, have now become a rarity with the use of the antituberculosis drugs.

Thoracoplasty was unsuccessful in 9 patients with multiple cavities or cavities in the middle or lower lobes. A small number with this type of pathologic condition were cured by operation, but the percentage of successful cases was so small that thoracoplasty would not have been considered as a method of treatment had some other procedure been available.

When infection of the wound occurred in the days before penicillin was available, the wound had to be opened widely and packed in order to control the infection; the second-stage operation could not be performed until the wound was well healed. This frequently took from six to eight weeks. Because of this delay, very little additional collapse was obtained after the second stage and cavities, therefore, ofttimes remained open. In our series, there were 7 patients in whom the second stage of the thoracoplasty had to be unduly delayed because of wound infection. Today this operative complication rarely, if ever, occurs, and a two-stage thoracoplasty can usually be completed in ten days. Furthermore, the various types of thoracoplasty which have been evolved in the past few years, using various methods of plombage in conjunction with rib resection or even leaving the ribs in situ, can be performed in one stage.

Six patients in our series were operated upon as a ''last resort.'' They were in poor general condition; they had extensive disease; and despite all measures available at the time, they could not be "built up" sufficiently to be regarded as good operative risks. Two of these patients are alive today, but they do have positive sputum. Both had had a destroyed lung, for which thoracoplasty, at that time, was the only procedure that could be used. They both live quite active lives, and we can assume that were it not for the benefits derived from the collapse obtained by thoracoplasty, they would have died before

DISCUSSION

One of the ordinary methods of reporting the results of surgical treatment is shown in Fig. 1. This gives the status of all patients on a specific date, the last follow-up date. This does not give the complete picture for (1) the results in cases with varying lengths of follow-up are considered with equal significance, and (2) temporary exacerbations with positive sputum are not discernible if the patient reconverted without additional surgery.

In analyzing the results of a chronic disease such as pulmonary tuberculosis, with its many periods of exacerbation and remission, we have tried to portray in Fig. 2 a year-by-year picture of the results. This method is also not perfect because it can only be used for those patients who remain alive. Those who died, either from tuberculosis or from a nontuberculous disease after being considered as cured, cannot be carried in the figures beyond the year of death.

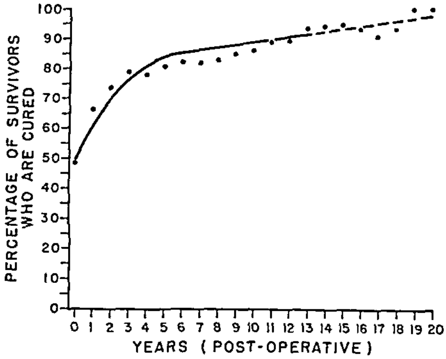

Fig. 2.

This then shows the status of only the living patients in each postoperative year. If a patient had only one positive sputum in a year, he was considered as "positive" in that year. A number of patients, although clinically well, have had an occasional report of a positive sputum over the years. Fig. 2 also gives expression to such incidents as the late conversion which occurred in 4 patients. In Fig. 1 they are considered as cured, but in Fig. 2 they are not classified as such until the year of persistent negative sputum.

Although neither of these charts gives the complete picture of the results, we believe that they supplement each other, thereby presenting a more accurate expression of what thoracoplasty accomplished in the treatment of pulmonary tuberculosis in the pre-antibiotic era.

SUMMARY

1. A complete analysis of thoracoplasties performed upon 180 patients at the Montefiore Hospital, New York City, from 1935 through 1948 is presented.

2. There has been a 100 per cent follow-up

sexes the ordinary clothing gave adequate concealment. Arm and shoulder mobility seldom was affected seriously. The occasional severe scoliosis occurred in the patients who developed wound infection. Today, as previously stated, this complication rarely ensues.

The study of those patients in whom cough and expectoration (with negative sputum) persisted revealed that considerable fibrosis and bronchiectasis was probably present before operation. This was visible on their preoperative roentgenograms. Today the antituberculosis drugs prepare the patient for operation so much more quickly that fibrosis and bronchiectasis are minimal.

From the few post-mortem examinations available to us on patients who died of other causes after cure of their tuberculosis, we can state that thoracoplasty did result in a pathologic, as well as a clinical, cure.

There has been no severe reduction in pulmonary reserve in any patient who had had uncomplicated unilateral pulmonary disease; and some of these patients are alive up to twenty years after thoracoplasty. One patient who had been well for seventeen years has developed cor pulmonale, but she had bilateral collapse therapy; thoracoplasty on the right, and oleothorax on the left. Cor pulmonale did develop and was the decisive cause of death in a few patients in whom the thoracoplasty was unsuccessful in preventing the progression and spread of the disease; but in those patients in whom a unilateral tuberculosis had been cured, there were no late deaths from cor pulmonale.

It is interesting to note that there were 4 patients in our series in whom the conversion of sputum did not occur before the eighth, ninth, tenth, and thirteenth postoperative years, respectively. When this conversion took place, it did so without the addition of any other form of surgical therapy and without the administration of any antituberculosis drugs. Curiously enough, once the sputum became negative in these late conversions, it remained so.

From our study, we can report that thoracoplasty for pulmonary tuberculosis performed in the pre-antibiotic era resulted in 73.9 per cent cures. These results are, in the main, similar to other reports which have recently appeared in the literature. We believe we are justified in postulating that a much higher percentage of good results would be obtained were a similar group of patients operated upon today with the aid of all the new drugs and techniques. And were we to eliminate from our present-day operations those patients with pathologic conditions which we now know are not amenable to collapse therapy, we believe our results would approximate those of resectional therapy.

It is significant that the ideal pathologic condition for thoracoplasty is also an ideal case for resectional therapy; and in our enthusiasm for the latter type of operation, thoracoplasty is rarely considered as a method of treatment. But it is also significant that as the follow-up period lengthens upon patients who have had resectional therapy, an increase in the number of reactivations has been observed. When thoracoplasty is performed only for suitable types of disease, it will yield a high cure rate; therefore, it should not be discarded from the surgical treatment of pulmonary tuberculosis. The other methods of surgical therapy will have to equal or better the percentage of cures after a prolonged follow-up period in order to justify their use in preference to thoracoplasty.

THE SURGICAL TREATMENT OF PULMONARY HEMORRHAGE IN TUBERCULOSIS

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PULMONARY hemorrhage is a serious complication of tuberculosis because it frequently causes spread of the disease. Hemorrhage which persists or recurs or is large in volume may be a threat to life itself. This paper concerns itself, in part, with the reporting of resectional surgery performed without complications in 3 cases in which the risk of operation was deemed desperate.

CASE REPORTS

The following 3 case histories summarize our recent surgical experiences with this problem. In 1952, one emergency pneumonectomy was performed. In 1954, one pneumonectomy and one right upper lobectomy were performed.

Case 1.—This 26 year old Negro man's first indication of pulmonary tuberculosis was a single hemoptysis of 50 c.c. of blood in January, 1951. Examination revealed a large left upper lobe cavity, and bilateral pneumonic infiltrations. The stained smear of the sputum was positive for tubercle bacilli. The patient received PAS and streptomycin daily for 18 months. During this period a left phrenic crush and pneumoperitoneum were performed, and the patient was kept at bed rest. He then left the hospital against advice.

Two months later, after 3 days of recurrent hemoptyses, he was re-admitted to the hospital. Examination disclosed findings similar to those found at the time of the first admission. Bleeding continued. On the sixteenth day after admission, following recurrent hemoptyses amounting to 3,300 c.c. in 5 days, and further involvement of the right lung, left pneumonectomy was performed. Chemotherapy consisted of isoniazid and PAS. The right lung infiltrations cleared slowly. The patient was discharged 9 months postoperatively, and chemotherapy was continued for a total of 22 months. Thereafter, the patient worked as a carpet layer.

Roentgenograms of March, 1954, and March, 1955, were identical in appearance and showed no evidence of tuberculous activity. Sputum cultures have been negative for more than 2 years.

This patient's x-ray studies *do* show a thin-walled "cyst" 2 cm. in diameter in the right middle lobe (Fig. 1). Its size changes with respiration. Attempts to demonstrate a focus of tuberculosis have been unsuccessful, even from cultures taken during and after hospitalization for pneumococcal pneumonia of the right middle lobe, March to May of 1955.

Pathologic examination of the resected left lung revealed two aneurysms of branches of the pulmonary artery in the walls of cavities. Bacteriologic studies showed complete sensitivity to streptomycin (no growth in medium containing 3.5 mcg. streptomycin per milliliter), despite 18 months of streptomycin and PAS given previously.

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3. The cases in which an unsatisfactory result was obtained have been analyzed in an attempt to determine the cause for the poor outcome.

4. Seventy-three and nine tenths per cent of the patients were cured by thoracoplasty alone. Surgical mortality was 5 per cent.

5. Some of the commonly held objections to thoracoplasty are discussed. They were not found to be prevalent in our cured group of patients.

6. The results which might be obtained by thoracoplasty, if performed today, are postulated.

7. Thoracoplasty performed upon a well-chosen group of patients should yield long-term follow-up results comparable to the results thus far obtained from resectional therapy, for which similar lengthy follow-up periods are not as yet available.

8. There are indications for the use of thoracoplasty in the treatment of pulmonary tuberculosis and this procedure should not be discarded.

9. The results are charted by two methods: (1) the end result on the last follow-up date, and (2) the status of the survivors in each postoperative year.

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Examination revealed a large right upper lobe cavity, caseous pneumonic processes in the right middle and lower lobes, and early infiltrates in the left lung (Fig 2) Recurrent hemoptyses and increased spread to the same and to the opposite lung led to right upper lobectomy with concomitant four rib thoracoplasty[1] one month after admission, on Sept 28, 1954 There were no pulmonary or pleural complications postoperatively Chemotherapy postoperatively was viomycin and PAS, discontinued because of an elevated BUN, followed by pyrazinamide and PAS The sputum became negative in November, 1954, 2 months after surgery

This patient was examined in November, 1955 Five sputum cultures were negative for acid fast bacilli A roentgenogram of the chest showed no evidence of active disease

The removed right upper lobe contained a large cavity filled with blood clots A specific bleeding site was not demonstrated In drug susceptibility tests, the tubercle bacilli from the sputum cultures on admission showed 4+ growth in media containing 15 mcg of streptomycin per milliliter There was 2+ growth in media with 200 mcg of streptomycin per milliliter[4]

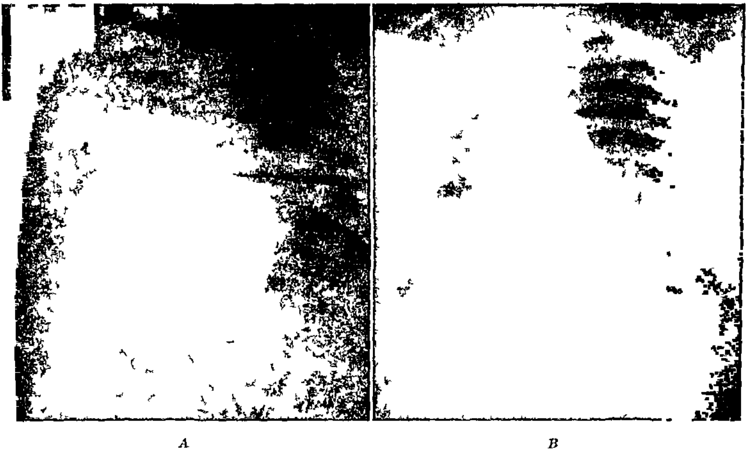

Fig 2 —Case 3 *A* Preoperative bedside roentgenogram showing extensive tuberculosis bilaterally

B Roentgenogram made 7 months after right upper lobectomy and partial thoracoplasty of ribs 2 3 4 and 5

COMMENT

The sequence in each case was inadequate and interrupted chemotherapy, when the patients left the hospital against advice On re admission, each patient was found to have a large, thick walled cavity There was positive sputum in large quantity, and there was reason to suspect resistance to previous forms of chemotherapy These patients appeared to have established the pattern of recurrent or persistent bleeding in large quantities, and had deteriorated to the point where medical therapy alone was judged to be inadequate to arrest bleeding and to preserve life

Review of Fatal Cases of Pulmonary Hemorrhage—The records and roentgenograms of 21 patients who died as a direct result of pulmonary hemorrhage in the 10 year period, 1945 to 1954, on the Bellevue Hospital Chest

CASE 2.—In December, 1952, a 22-year-old Negro man was admitted to Bellevue Hospital Chest Service for treatment of pulmonary tuberculosis. Examination showed a left upper lobe cavity measuring 6 cm. in diameter, and diffuse involvement of the left lung and of the upper lobe of the right lung. Streptomycin and PAS were given daily for 3 months when the patient left the hospital against advice. He was re-admitted with a single small hemoptysis one month later, but stayed only one day. Nine weeks later he was re-admitted without hemoptysis. He was transferred to a sanatorium for treatment, and received streptomycin, isoniazid, and PAS. He was discharged 11 months later, April 1954, without further medication.

He was admitted to the hospital again on June 10, 1954, with a history of an hemoptysis of 200 c.c. This had been preceded by 2 weeks of cough and fever. Large cavities were present in each lobe of the left lung. In the next 14 days, bleeding totalled 1,300 c.c. There was spread to the right lung. Left pneumonectomy was performed on June 24, 1954, 17 months after the first admission for tuberculosis. The pneumonia in the right lung cleared rapidly. He was treated with streptomycin, isoniazid, and PAS. Sputum studies in August, 1954, were negative. The patient left the hospital against advice in September, 1954.

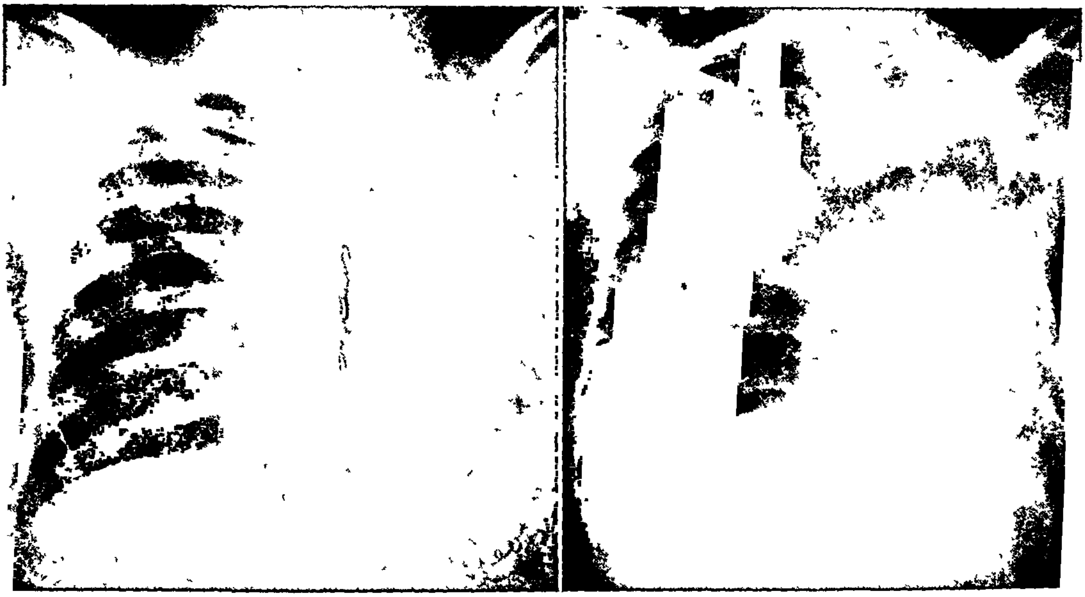
A. B.

Fig. 1.—Case 1. *A*, Preoperative roentgenogram in August, 1952, illustrates the extent of bilateral tuberculosis.
B, Postoperative roentgenogram in May, 1955, shows right middle lobe "cyst."

This patient has continued to have sputum negative for acid-fast bacilli on smear and culture. When last seen in May, 1956, he was well, and the x-ray examination of his chest showed no new or active disease.

The removed lung revealed large cavities in each lobe and erosion through the wall of a branch of the pulmonary artery to the left upper lobe. There is no record of sensitivity studies on the tubercle bacilli from this patient.

CASE 3.—A 47-year-old Negro man entered the Bellevue Hospital Chest Service in August, 1954, following 3 weeks of hemoptysis. In the first 2 weeks, the sputum was blood streaked; in the last week there was hemoptysis of 300 c.c. a day. He was known to have had tuberculosis for one year. He had been treated in another hospital with streptomycin, isoniazid, and PAS daily for 5 months, and with an additional 3 months of isoniazid alone before leaving that hospital against advice. A 2-month interval without therapy and at full activity, including full-time employment as a store clerk and messenger, preceded this admission to Bellevue Hospital.

the use of barbiturates and 15 or 30 mg. doses of codeine every few hours if necessary. Heavy sedation or narcosis is avoided. Oxygen by tent or nasal catheter is used if necessary. Blood is typed and cross-matched. The patient's response to therapy is observed and noted.

Pneumothorax has been attempted, with the hope of arresting bleeding by changing the dynamics in the cavity. It was not successful when tried in several of the 21 cases cited, because of pleural symphysis. This experience presumably explains the infrequent attempts to employ pneumothorax in this group.

Two patients were treated for hemorrhage with thoracoplasty. In one of these the hemorrhage stopped after the thoracoplasty had been performed. Pneumonectomy was performed 2 years later for a destroyed lung. In the other, the hemorrhage finally was controlled after thoracoplasty and revision. This patient died one year later as a consequence of diffuse bilateral spread of tuberculosis and subsequent fatal hemorrhage (Fig. 5).

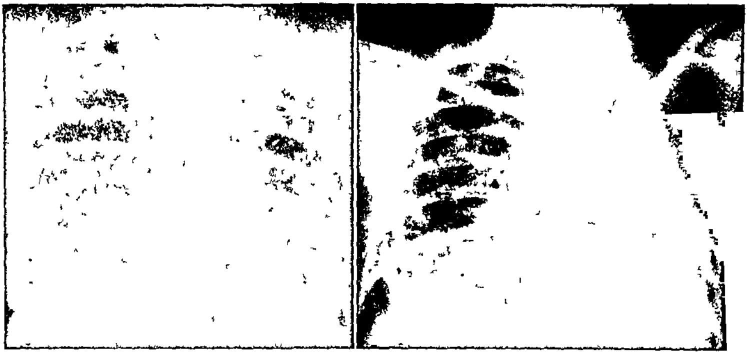
A. *B*

Fig. 5.—*A*, Admission roentgenogram of 35-year-old patient, whose subsequent pulmonary hemorrhage was controlled by (*B*) two-stage thoracoplasty. Death occurred one year later from hemorrhage from cavity in superior segment of right lower lobe

Indications for Resectional Surgery.—It is of interest to attempt to define the indications for resectional surgery in pulmonary tuberculosis complicated by hemorrhage. From the experience presented, the indication for resectional surgery is limited to the instance of the patient with the thick-walled cavity who continues to have pulmonary hemorrhage in large amounts, and in whom a fatal outcome seems certain without operation.

The decision to operate and the timing of surgery are difficult, and may be determined only after repeated observations of the patient as hemorrhage continues. In order to consider excisional surgery, the cavitary disease must be unilateral. The contralateral disease must be recent.

In the 3 patients upon whom a decision to operate was made, it was known that there was unilateral cavitary disease. The episode of bleeding consisted of three or more large hemoptyses. There was spread to the other side. There

Service were reviewed (Fig. 3). One exsanguinated, 10 died of asphyxia, and 10 died of toxicity and hypercapnea following spread of tuberculosis (Fig. 4).

The facts indicated that age and sex were of no significance. The known duration of tuberculosis in these patients had no bearing on the quantity or frequency or outcome of the hemorrhage. The history of previous hemorrhage had no relation to the severity of the current hemorrhage.

The local characteristics of the lesions appeared to be more important. All patients had cavities, which were of various sizes, and located in upper or lower lobes, or both. All these patients had cavities with thick walls.

Thus, it would appear that the setting for continuing hemorrhage is to be found in the patient who has a thick-walled cavity.

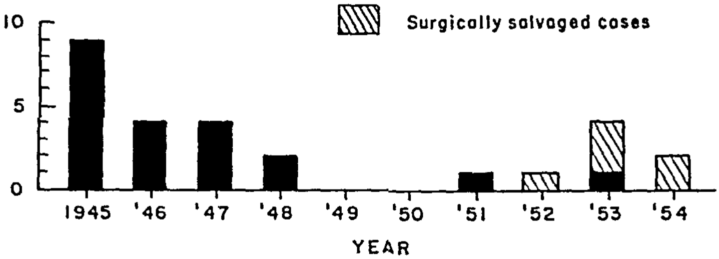

Fig. 3.—The incidence of deaths from pulmonary hemorrhage on the Belevue Hospital Chest Service, 1945 to 1954, with indication of patients saved by surgery.

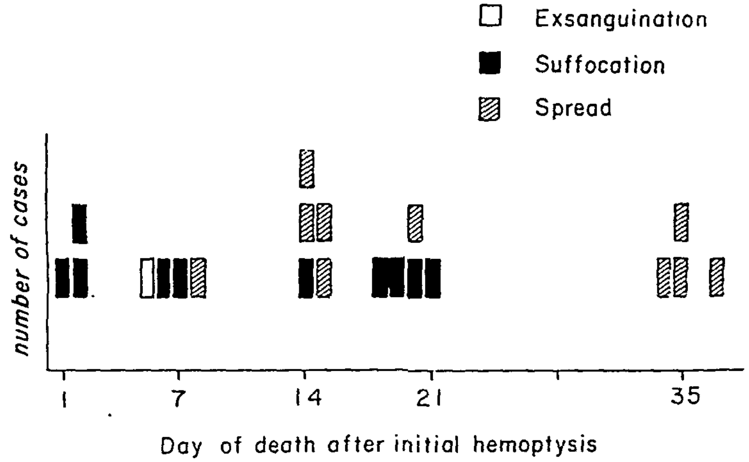

Fig. 4.—The causes and time of death from pulmonary hemorrhage in 21 patients, 1945 to 1954.

DISCUSSION

Therapy of the Patient With Hemoptysis.—We have no statistics of the degree and severity of hemorrhage in patients who were treated successfully by therapy other than surgery. Certainly the majority of pulmonary hemorrhages cease spontaneously or respond to nonsurgical therapy after a short period of time. This is to be explained convincingly to patients with pulmonary hemorrhage upon admission.

The patient is positioned with the bleeding side dependent, and the head of the bed lowered, to aid cleansing the tracheobronchial tree while limiting gravitational spread. Coughing is permitted and encouraged at intervals. Excessive coughing is to be avoided mainly by patient cooperation, but with

CLINICAL MANAGEMENT OF POSTTRAUMATIC RUPTURE OF THE THORACIC AORTA

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FOLLOWING severe nonpenetrating thoracic injury, the aorta may be torn, producing either a complete laceration with immediate exsanguination, or an incomplete tear of the vessel wall with delayed rupture or the subsequent formation of an aneurysm. Although reports of such lesions have appeared sporadically for many years,[1-9] only recently has there been much clinical interest devoted to them, due no doubt to their increasing frequency as accidents incident to high speed transportation become more common, and to advances in thoracic and vascular surgery which now afford a reasonable therapeutic approach to their clinical management.[10-20]

The purpose of this report is to describe 5 cases of thoracic aortic rupture due to nonpenetrating trauma, all of which were diagnosed clinically, and each of which represents one phase in the clinical course and management of this problem. On the basis of these and other reported experiences, a clinical approach to these lesions is suggested.

CASE REPORTS

Case 1.—T. C., a 42 year old man, suffered severe injury to the chest in an automobile accident in 1940, from which he recovered without incident. Upon his entrance into the army in 1941, a roentgenogram disclosed a mass in the left chest (Fig. 1, *A*) which was present also in 1944 and 1948 (Fig. 1, *B*). Retrograde aortography in March, 1957, confirmed the presence of an aneurysm in the descending thoracic aorta immediately below the left subclavian artery (Fig. 1, *C*, and *D*) which has remained constant in size since first visualized in 1941. This patient is entirely asymptomatic and is being followed by roentgenograms of the chest at 6 month intervals.

Summary. This posttraumatic thoracic aortic aneurysm located immediately below the left subclavian artery has been followed without evidence of its progression for 16 years.

Case 2.—J. H. F., a 24 year old man, was in an automobile accident in November, 1951. In 1953, upon discharge from the army, he was told that there was an abnormal shadow observed in the roentgenogram of his chest. He was first seen at the Denver VA Hospital in March, 1954, and has been followed at 6 month intervals ever since (Fig. 2). He remains asymptomatic and there is no evidence that the aneurysm has enlarged during this period.

Summary. This posttraumatic thoracic aneurysm was first observed 2 years after injury and raised the clinical problem of its resection versus the advisability of watchful waiting. The latter method was chosen and the lesion has remained unchanged for 4 years.

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was increasing respiratory distress. The patients appeared weaker and more ill in every way. In the desperate situations in which these judgments for resectional surgery have been made, the question of whether the contralateral spread was tuberculosis or blood pneumonia was not of sufficient importance to deter surgical intervention.

Individual case reports of pneumonectomy for the control of pulmonary hemorrhage in tuberculosis, by Ryan and Lineberry[3] and Ross,[2] applied similar criteria of selection and timing for excisional surgery.

Contraindications: We do not advocate excisional surgery in pulmonary hemorrhage except as a lifesaving measure to protect a patient from asphyxia and death. Although the coincident removal of the major focus of disease in each instance was a major dividend, we believe that properly timed elective resections will yield the best results.

It is conceded that advanced bilateral caseocavitary tuberculosis, pulmonary insufficiency, and other usual contraindications will obviate attempts at surgical therapy in many instances.

The Operation.—At each operation an experienced anesthetist gave a rapid, deliberate induction and employed a cuffed Magill endotracheal tube. The usual lateral decubitus position of the patient was employed with the head of the table lowered. It was felt that the theoretical advantages of a bronchial block or a divided, selectively occluding, endotracheal tube, or of an unusual position of the patient on the operating table, were unnecessary. Surgical technique was directed toward rapid occlusion of the bronchus to the involved lobe or lung.

SUMMARY

Most instances of pulmonary hemorrhage secondary to tuberculosis cease with proper medical therapy.

The setting for recurrent and uncontrollable hemorrhage is to be found in the patient who has a thick-walled cavity.

A report of one lobectomy and two pneumonectomies for the control of pulmonary hemorrhage, in the presence of thick-walled cavities, has been presented.

Twenty-one instances of fatal pulmonary hemorrhage have been reviewed. Seven patients might have been saved by pulmonary resection on the basis of current surgical considerations.

Medical, surgical, and anesthetic considerations in the patients with pulmonary tuberculosis and hemorrhage have been discussed.

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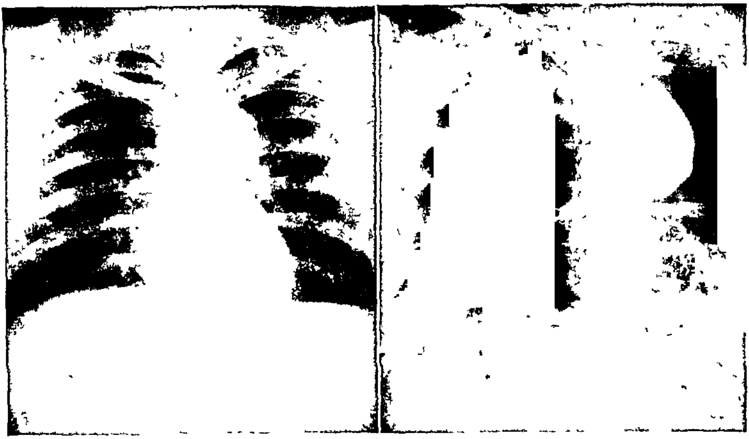


A. *B.*

Fig. 2.—Case 2. *A*, (1954). Two and one-half years following injury showing aortic aneurysm. *B*, (1955). Transcervical aortogram showing aneurysm unchanged.

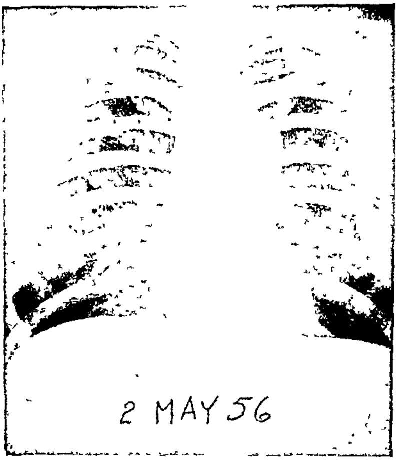


Fig. 2*C*.—Case 2 (1956). Four and one-half years following injury. Size of aneurysm unchanged.

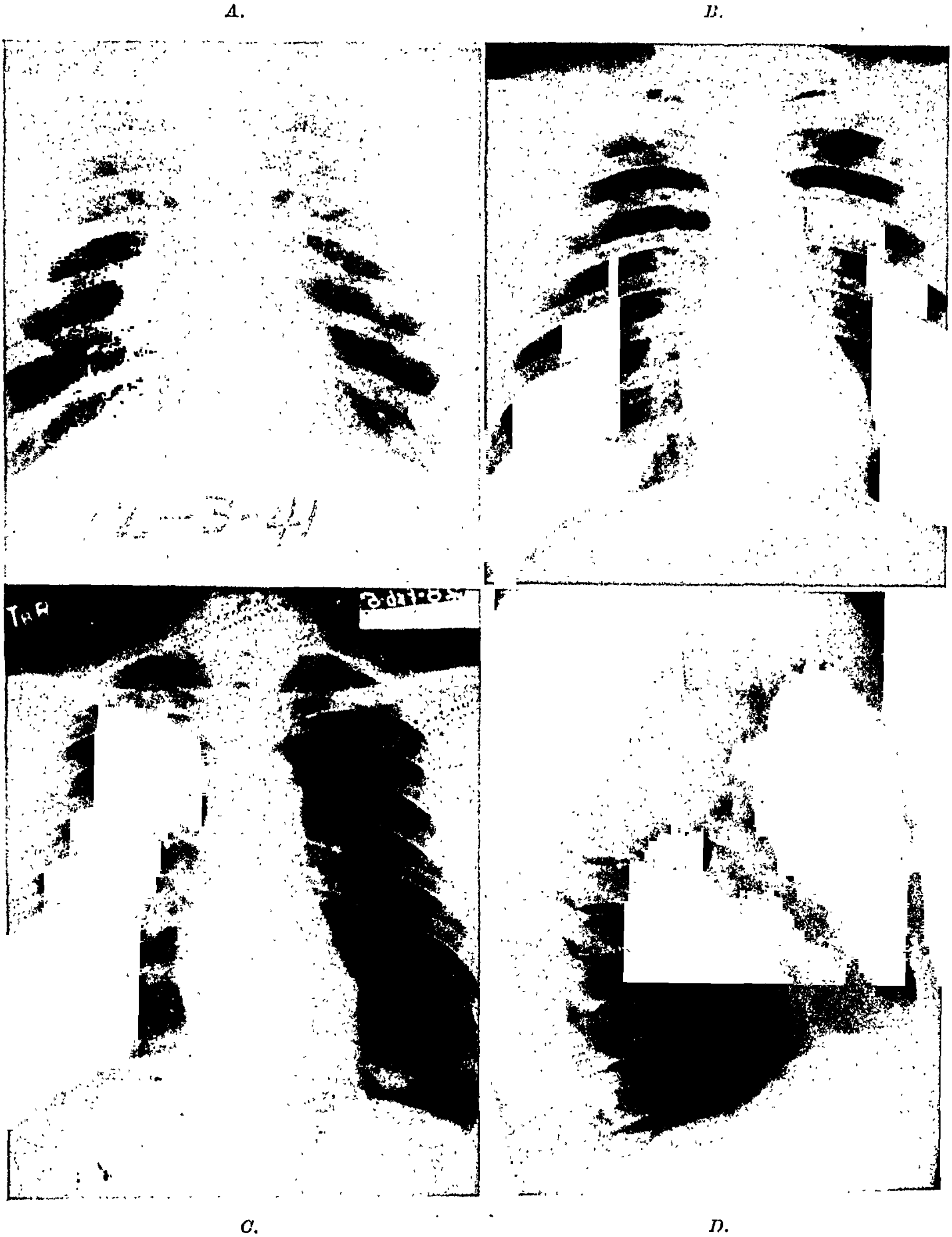


Fig. 1.—Case 1. *A*, (1941). One year following trauma showing slight widening of aortic arch.

B, (1948). Seven and one-half years folowing trauma. Aneurysm unchanged in size.

C and *D*, (1957). Posteroanterior and lateral chest x-rays 16 years following injury. No enlargement but marked calcification of the aneurysm.

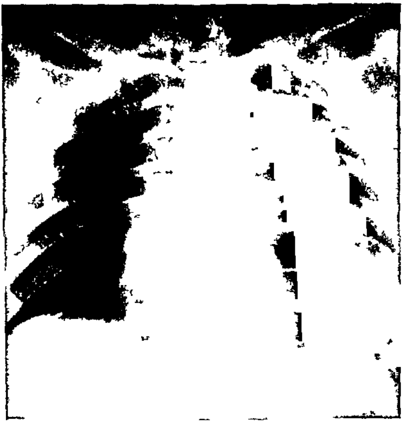


Fig 4

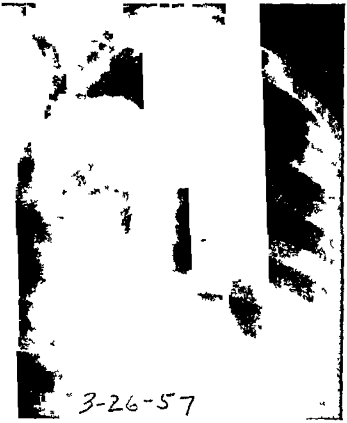


Fig 5

Fig 4—Case 4 Twelve hours after fall from third story window There is marked mediastinal widening

Fig 5—Case 4 Three weeks following injury The angiocardiogram shows the aortic aneurysm that was not present at thoracotomy immediately following injury

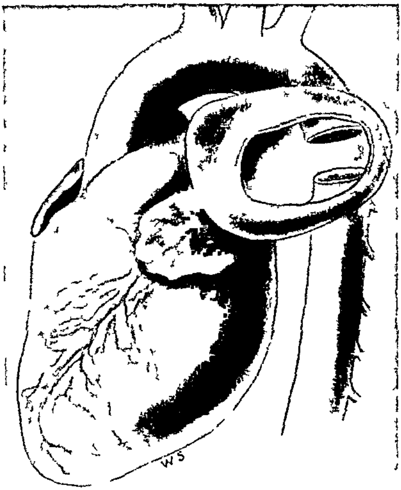


Fig 6—Case 4 Illustration of findings at second operation The normal aortic wall has been circumferentially transected through the intima and media forming an aneurysm densely adherent to the left pulmonary artery main stem bronchus and recurrent laryngeal nerve

CASE 3.—P. A., a 20-year-old man, suffered a fractured right olecranon and multiple lacerations of the face in an automobile accident on Jan. 1, 1957. He was hospitalized elsewhere and because of some anterior chest pain was thought to have a "possible fracture of the sternum." He was admitted to the Denver VA Hospital 2 days after injury, at which time his condition was good, although a roentgenogram of the chest (Fig. 3) showed questionable mediastinal widening and elevation of both diaphragms, patchy atelectasis, and minimal fluid in the left base. The olecranon fracture was repaired under general anesthesia without incident on the sixth postinjury day.

He was about to be discharged on the ninth postinjury day (Jan. 10, 1957), when he complained of moderate pain in his back and upper chest. His vital signs began to deteriorate later in the day, at which time breath sounds were found to be absent in the left chest. Tubes were inserted into the chest, 1,000 c.c. of old blood removed, and his condition improved markedly. Two hours later his condition deteriorated, femoral pulses disappeared and he complained of numbness in his feet. As he was being taken to surgery (approximately 8 hours after his complaint of back pain) he suddenly went into shock. An emergency left thoracotomy revealed a ruptured thoracic aortic aneurysm. The bleeding was stopped by manual pressure over the torn aneurysm, and the heart resuscitated sufficiently to allow a formal thoracotomy. The aneurysm was resected and a nylon graft inserted, but the patient died shortly after the occluding aortic clamps were removed.

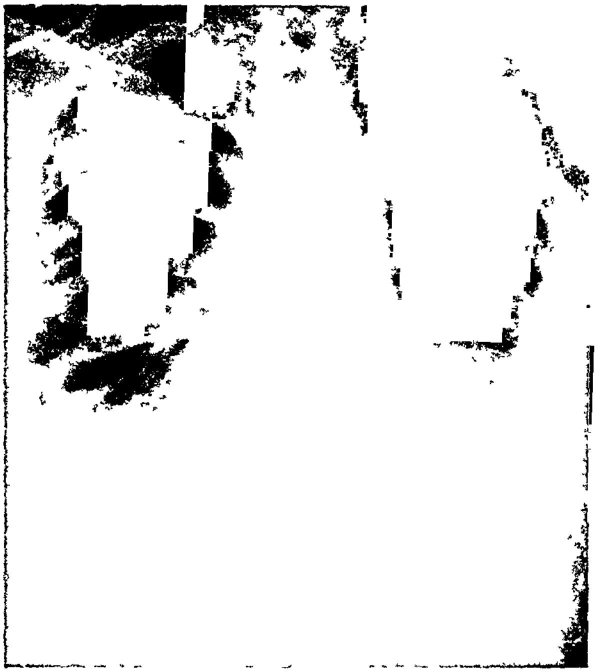


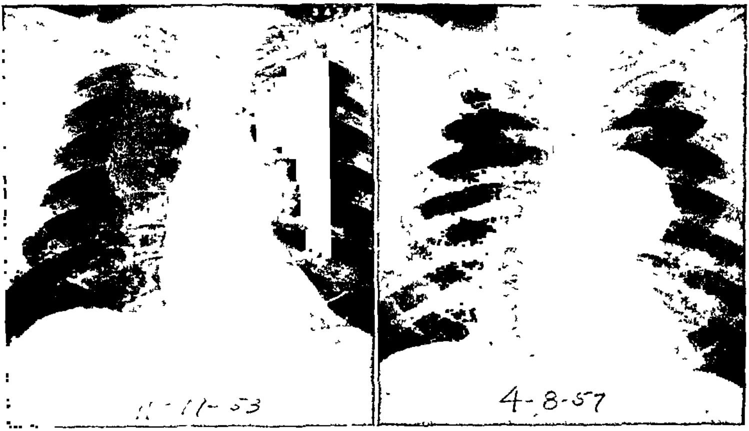
Fig. 3.—Case 3 Three days after trauma. Showing diffuse mediastinal widening and fluid at both bases but predominantly on the left

Summary: For 9 days following injury this patient had essentially no symptoms referable to the chest. Within an 8-hour period, however, he developed chest pain, hemothorax, and ultimately went into shock at the moment when the aneurysm ruptured into the left pleural space. Heroic operative measures were too late for subsequent recovery.

CASE 4.—R. V. A., a 44-year-old man, fell from a third story window during an epileptic seizure and struck his left chest on the ground, resulting in multiple rib and mandibular fractures. Upon entry to the Denver VA Hospital on Feb. 19, 1957, a roentgenogram of the chest showed a left pneumothorax and marked mediastinal widening (Fig. 4). The femoral pulses fluctuated in intensity but his general condition was satisfactory. A diagnosis

Summary: Thirteen years following chest injury an aneurysm of the descending thoracic aorta was thought to be enlarging and producing back and chest pain localized to this region. It was successfully excised and replaced with a homograft, furnishing relief of symptoms.

CASE 6.—W. B. L., a 63 year old man, suffered severe trauma to the left chest at the age of 17 when he was kicked by a mule. A mass was noted in the descending portion of the aortic arch in 1948, and roentgenograms of the chest showed gradual enlargement in 1953 (Fig. 8, *A*). He was first seen at the Denver VA Hospital in 1957, at which time the aortic aneurysm had appreciably enlarged (Fig. 8, *B*). Repeated analyses of the blood and cerebrospinal fluid were negative for syphilis. Because of advanced metastatic carcinoma of the bladder no direct attack on the aortic aneurysm has been advised.



A. *B.*

FIG 8—Case 6. *A*, (1953). Forty-three years following mule kick to left chest. *B*, (1957). Four years later and 47 years following injury showing marked growth in size of the aneurysm.

Summary: This descending aortic aneurysm is most likely due to severe trauma to the chest suffered 47 years ago. Proof of such etiology is, however, lacking. It has been followed by serial roentgenograms of the chest for 9 years and has shown marked enlargement during the past 4 years (43 years following injury).

The salient features of these 6 cases are summarized in Table I.

DISCUSSION

The number of cases of posttraumatic thoracic aortic aneurysms that have now been reported removes this lesion from the realm of curiosity to one whose natural history should be carefully described and its proper clinical management thereby delineated. The characteristic trauma producing these lesions and the hydrostatic principles involved have been well described elsewhere[20, 21] and need not be mentioned further.

of thoracic aortic rupture was made and a left exploratory thoracotomy performed within a few hours after his admission to the hospital. Although there was diffuse ecchymosis surrounding the entire length of the thoracic aorta, no site of bleeding could be found. The aorta immediately below the left subclavian artery was minutely inspected, but no evidence of laceration was found. As the postoperative serosanguineous fluid finally cleared from the left chest, an aortic aneurysm was noted immediately beneath the left subclavian artery. This was first visualized on March 10 (Fig. 5), 21 days after injury and subsequent negative exploratory thoracotomy. Bronchograms the following day showed that the mass occluded the left upper lobe bronchus. At operation on April 3, 1957, under hypothermia anesthesia (29° C.), the aorta was cross-clamped below the left subclavian artery and the aneurysm opened. There was found to be a 360 degree circumferential, intimal tear with a 6 cm. separation of aortic wall opening into the aneurysmal sac (Fig. 6). The aortic wall above and below the aneurysm appeared normal. Twenty minutes after the aorta was clamped, as a lyophilized aortic graft was being inserted to bridge the defect, totally resistant ventricular fibrillation suddenly developed and the patient died.

Summary: Following severe injury to the chest, roentgenograms demonstrated widening of the mediastinum. Exploratory thoracotomy revealed diffuse periaortic hematoma, but *despite close inspection of the aorta no evidence of rupture was found. Three weeks* following injury and negative thoracotomy an aortic aneurysm was demonstrated beneath the left subclavian artery.

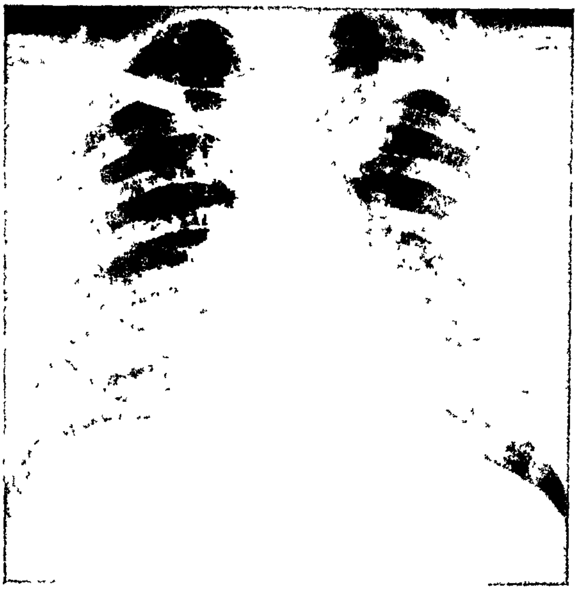


Fig. 7.—Case 5. Fifteen years after severe trauma showing aortic aneurysm involving the lower portion of the descending aorta. This lesion was successfully excised and replaced with a lyophilized homograft when it began to enlarge and cause symptoms.

CASE 5.—C. M., a 53-year old man, was in 2 automobile accidents, one in 1943 and another 1944. In both accidents he suffered severe injuries to the left chest and flank and had had broken ribs on this side. In 1953, a mass was noted in the left chest on a routine roentgenogram. He was asymptomatic, except for some low lumbar back pain, until 1956 when he began to notice pain in the left lower chest and back. X-ray examination of the chest (Fig. 7) showed a questionable enlargement of the mass compared to previous studies. May 21, 1956 (at the Weld County General Hospital, Greeley, Colo.), the patient was operated upon under hypothermic (28° C.) anesthesia and a lemon-shaped aneurysm overlying the seventh thoracic vertebra and measuring 11 cm. in greatest diameter was removed. A lyophilized aortic homograft was used for replacement. The patient's postoperative course was uneventful. Subsequently, the back pain disappeared, and he is gainfully employed.

TABLE II. SUMMARY OF CASES PREVIOUSLY REPORTED

AUTHOR	NO. CASES	TIME FROM INJURY TO DIAGNOSIS	REMARKS
Kemp (1923)[1]	1	Immediate	Tear, ascending aorta; post-mortem diagnosis
Kuhn (1925)[2]	1	10 days	Post-mortem diagnosis
Corbitt and Mathews (1937)[3]	1	20 hours	Post-mortem diagnosis
Kleinsasser (1943)[4]	1	Immediate	Post-mortem diagnosis
Forbes (1944)[5]	1	30 hours	Post-mortem diagnosis
Strassman (1947)[6]	69	1-18 hours	Review of 7,000 autopsies
Henning and Agmar (1948)[7]	1	48 hours	Post-mortem diagnosis
Bradford and Johnston (1950)[8]	1	5 weeks	90 cases reviewed; 7 survived over 18 hours
Rice and Wittstruck (1951)[9]	1	20 days	Female at term pregnancy
Hollingsworth et al. (1952)[10]	4	4 months 2 years, 4 months 1 year 2 months	1-7 year follow-up; 2 cases with thoracotomy only; 1 case had Cellophane wrapping of aneurysm
Wyman (1953)[11]	3	8 days 15 hours 11 hours	Post-mortem diagnoses; all were aneurysms of posterior aortic arch
Kastl (1953)[12]	1	10 days	Post-mortem diagnosis
Bahnson (1953)[13]	1	8 months	Saccular aneurysm; excision with closure
Goyette et al. (1954)[14]	3	2 years 8 years 4 months	Thoracotomy; no attempted repair; no surgery because of location; excision; died 3 months later from rupture
Bornstein (1954)[15]	1	4 hours, 10 minutes	Chest x-ray 3 hours posttrauma said to be negative; post-mortem diagnosis
Hardin et al. (1954)[16]	1	2 years	Increase in size over 8-year period; excision and graft; hypothermia; operative death from ventricular fibrillation
Stranahan et al. (1955)[17]	1	5 years	Successful resection and homograft replacement; temporary by-pass shunt
Sarot and Lazzarini (1955)[18]	1	14 years	Successful resection and homograft replacement; permanent by-pass shunt
Cooley and DeBakey (1955)[19]	1	8 years	Tangential aneurysmectomy; later excision and graft; successful
Zehnder (1956)[20]	1	4½ days	Post-mortem diagnosis

TABLE I. SUMMARY OF CASES IN THIS SERIES

PATIENT	TIME INTERVAL INJURY TO DIAGNOSIS	REMARKS
J. H. F.	3½ years	No surgery; 5½ year follow-up; no increase in size of aneurysm
P. A.	10 days	Emergency surgery; excision with attempted graft replacement; died
R. Van A.	16 hours	Immediate thoracotomy with essentially negative findings; surgery 40 days posttrauma for aneurysm; attempted excision; death from ventricular fibrillation
T. C.	16½ years	No surgery; calcification of aneurysm but *no increase in size*
C. M.	13 years	Surgery with excision and homograft replacement; hypothermia; no neurologic sequelae
W. B. L.	43 years	Followed for 9 years with marked enlargement of aneurysm for last 4 years; no surgery because of inoperable bladder carcinoma

Natural History of the Lesion.—Complete laceration of all layers of the aorta by nonpenetrating chest trauma follows extremely severe injury and results in almost instantaneously fatal exsanguination. Although such lesions are common findings at the coroner's inquest following airplane accidents,[21] they have little significance for the clinician. In the future the proper use of seat belts and other devices may reduce the number of such immediately fatal injuries.

Following incomplete severance of the aortic wall, on the other hand, there is a varying period of time before delayed perforation may develop or before an aneurysm may form. Their proper clinical management depends on the developmental course of the untreated lesion, which heretofore has not been emphasized.

More than one half of the aortic tears following blunt thoracic trauma are located immediately below the left subclavian artery,[6] the remaining being either at the origin of the aorta from the heart, on the concavity of the arch, or in the descending aorta. Commonly the tears are transverse and occasionally are completely circumferential. The intima is torn but with a varying thickness of the adventitia and media remaining intact. External examination of such lesions immediately following injury reveals (Case 4) only a diffuse subadventitial ecchymosis along the aorta, without any localizing collections of blood or bulging of the vessel wall.

The time that the unbuttressed aortic adventitia can withstand arterial pressure before it ruptures, or an aneurysmal dilation occurs, undoubtedly depends upon the depth and extent of the original internal laceration. The adventitia accounts for 60 per cent of the tensile strength of an artery[22] but will ultimately give way if the other layers are disrupted. The significant fact is that such a latent period exists following injury, during which time the lesion may be overlooked by the unsuspecting clinician. On the other hand, if the progressive nature of the lesion is recognized, an aggressive surgical approach during this period may be of clinical benefit in avoiding aortic rupture. The time between injury and aortic rupture in Case 3 was 9 days. The previously reported experience in this regard is summarized in Table II and illustrates that

Should no such laceration be found to account for the periaortic hematoma, the question arises as to the advisability of an exploratory aortotomy performed between occluding clamps immediately below the subclavian artery so as to visualize the posterior wall of the aorta at this point. In the case cited, such a maneuver would in all likelihood have saved the patient's life.

A small incomplete aortic laceration found in the immediate posttrauma period, before aneurysmal dilatation has developed could probably be successfully repaired by simple vascular suture without the necessity for prolonged aortic occlusion with resection or grafting. Since such lacerations characteristically appear in the posterior aspect of the aorta, adequate exposure may be difficult.

The clinician must remain vigilant for signs of aortic rupture for at least 4 weeks following injury and must not ignore such a catastrophic possibility merely because of several asymptomatic days or even weeks. Normally there will be a warning period of a few hours, during which time the alert and aggressive surgeon will have time to perform thoracotomy before massive hemorrhage supervenes.

Finally, the clinician may be faced with the problem of an asymptomatic aneurysm discovered months or even years following injury. Since there is good evidence that some of these lesions may remain unchanged for many years, such aneurysms probably can safely be followed at 6-month intervals by serial roentgenograms or laminograms. Should there be incontrovertible evidence of enlargement, or should the lesion become symptomatic, excision and graft replacement can be employed.

Hypothermia (to 29 to 30° C.) and some by-pass shunt around the aneurysm should be used, both to minimize the likelihood of spinal cord damage and to relieve the heart of the burden of working against an occluded thoracic aorta. The hypothermic heart tolerates poorly such outflow resistance unless inflow occlusion is simultaneously employed.[24]

SUMMARY

1. Six clinical cases of posttraumatic thoracic aortic aneurysm have been described which represent various phases of the natural history of this condition.

2. On this basis and on the previous experience of others, the following clinical approach is suggested:

a. Early thoracotomy should be performed if incomplete aortic laceration is seriously suspected. Such a procedure should include careful inspection and palpation of the entire length and circumference of the thoracic aorta for evidence of laceration. Exploratory aortotomy may even be necessary to visualize the site of rupture if no other method discloses the origin of diffuse periaortic ecchymosis.

b. An asymptomatic period of 2 to 3 weeks may elapse following severe chest injury before delayed aortic rupture occurs.

c. Immediately prior to the exsanguinating hemorrhage from an incomplete aortic laceration or from the resulting aneurysm, there commonly is a short essentially all such episodes occur during the first 3 weeks following injury. Although there may be little or no clinical evidence of aortic damage during most of this latent period, a short premonitory period usually precedes perforation and exsanguination. Like its atherosclerotic and syphilitic counterparts the traumatized aorta characteristically begins to leak slowly at first, producing symptoms of hemothorax, respiratory distress secondary to collapse of the left lung, and evidence of progressive blood loss. This pre-exsanguination period is the surgeon's last chance to staunch the flow of blood from the crumbling aneurysm wall, which like a distintegrating dam may start with a trickle but soon ends in a disastrous flood. The period from the first signs of perforation to death may be but a few minutes or as long as 24 to 48 hours.

Early aortic rupture following trauma characteristically occurs within the first 3 to 4 weeks. Thereafter an aneurysm may develop which, appearing as they do in an otherwise normal aorta, have a better prognosis than their syphilitic or atherosclerotic counterparts. Whereas most of the latter rupture within 8 months after onset of symptoms,[23] there is increasing evidence that many posttraumatic aneurysms found one month or more after injury remain indefinitely unchanged.

Clinical Management.—An immediate free aortic perforation or transection presents no clinical problem inasmuch as the casualty seldom survives to receive even the most preliminary medical attention.

When an incomplete aortic tear occurs following nonpenetrating trauma the clinical problem resolves itself into: (1) is there actually an aortic injury, (2) will the aorta perforate, (3) is an aneurysm developing, and (4) if an aneurysm is present, is it expanding or static and should therefore be excised?

A high index of suspicion for aortic injuries following severe nonpenetrating trauma to the chest is essential for their early recognition. Numerous case reports in the literature, in which the diagnosis was unsuspected until postmortem examination, testify to the previous ignorance of this lesion. Once aortic damage is suspected, examination of the chest must be done to determine questionable widening of the mediastinal shadow and to delineate the outline of the aorta. Vital signs should be recorded at frequent intervals for evidence of major blood loss and suitable measures provided for replacement therapy. Progressive widening of the mediastinal shadow, signs of blood loss, or unexplained left hemothorax necessitate exploratory thoracotomy at the earliest feasible moment, while the patient's general condition remains satisfactory.

The large number of reported cases that have been clinically unsuspected and treated by nonoperative means for hours or even days following injury, while they bleed fatally into the chest, make early thoracotomy mandatory if there is serious possibility of aortic rupture.

An incision in the left fourth interspace will allow adequate exposure of the entire aorta and an optimal approach to the area immediately inferior to the subclavian artery where most of the lacerations will occur. If there is any evidence of periaortic ecchymosis (as in Case 4), the entire thoracic aorta must be both minutely inspected and palpated for evidence of an occult tear.

TRANSFIXION OF THE AORTA ACCOMPANIED BY A BROWN-SÉQUARD SYNDROME

A Case Report

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THERE are few reports of successful suturing of lacerations of the thoracic aorta and survival is rare. The first successful suturing of an aortic laceration was by Dshanelidze,[4] in 1922. His patient was a 20-year-old man with a stab wound to the left of the sternum in the second interspace. The pericardium was opened and an 8 mm. wound about one centimeter from the heart was closed. In America, Blalock[2] reported the first successful suture of a similar wound, in 1932. His patient was an 18-year-old man with an ice pick laceration through the first left interspace and the intrapericardial portion of the ascending aorta. In 1941, Elkin[5] reported 2 cases of ice pick perforation of the ascending aorta in which operation was performed with one survival. Again, in 1944,[6] he presented a case of a stab wound of the intrapericardial aorta with survival after suturing. Beattie and Greer[1] reported what they believed was the fifth successful repair of a lacerated thoracic aorta. Their patient, a 21-year-old man, had been stabbed in the second left interspace and forty-eight hours later a 6 cm. portion of knife blade was removed from the ascending aorta within the pericardium. The 5 reported successful cases all concerned the intrapericardial ascending aorta and were remarkably similar wounds.

Significantly, in 1950, Bradford and Johnston[3] reviewed the literature and reported 90 cases of traumatic rupture of the aorta with only 4 patients surviving the injury. All of these survivors had sustained the aortic tear within the pericardium. Worthy of mention are 2 recorded cases of arteriovenous fistula between the aortic arch and the left innominate vein following stab wounds. The first successful closure of such a fistula was by Proctor in 1950.[8] His patient was a 25-year-old railroad employee who was struck by flying glass just above the left sternoclavicular joint and sustained a minor laceration. He presented himself a year later with cardiac failure and "buzzing" in his head. An almost exact duplicate of this case was reported by McCook.[7] His patient was stabbed in the same location and developed an identical fistula which was successfully closed.

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(1 to 36 hour) period of a lesser leak. This premonitory warning must be recognized instantly and immediate thoracotomy performed for operative cure.

d. Asymptomatic posttraumatic thoracic aortic aneurysms discovered incidentally more than a month following injury probably will stabilize and can be managed expectantly with serial roentgenograms of the chest at 6-month intervals. Should there be any evidence of enlargement or development of symptoms they should be excised and replaced with a suitable graft.

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tear was seen. It was then opened and the spinal cord found to be slightly edematous and congested. A No. 14 rubber catheter was passed upward and downward along the cord for a distance of three or four inches without encountering a block. The knife blade could not be found. A thoracotomy was then carried out through the ninth left interspace. The diaphragm was partially opened to facilitate exposure. The point of the knife was then easily palpated within the thoracic aorta at the level of the eleventh thoracic vertebra about ⅛ of an inch from the anterior wall in the midline of the vessel. A retropleural hematoma was present around the aorta posteriorly from the diaphragm to about the ninth thoracic vertebra and consisted of approximately 200 c.c. of blood. The aorta was then mobilized, occluded above and below the point of entrance with Crafoord clamps, and lifted off the knife blade anteriorly. The laceration was 1.5 cm. long and was closed with a continuous suture of No. 4-0 arterial silk. The time of total occlusion of the aorta was seven minutes. Various attempts to manipulate the protruding knife blade were unsuccessful since it was firmly imbedded in the vertebral body. Finally it was broken off flush with the vertebral body and the remainder left in situ. The removed portions are shown in Fig. 1. The chest was closed with a large drainage tube connected to a water-seal bottle. The patient received a total of 4,500 c.c. of blood during these procedures which he tolerated very well. Postoperative x-rays revealed that the laminectomy was one vertebra too high, but even so it is believed that the blade remained extradural.

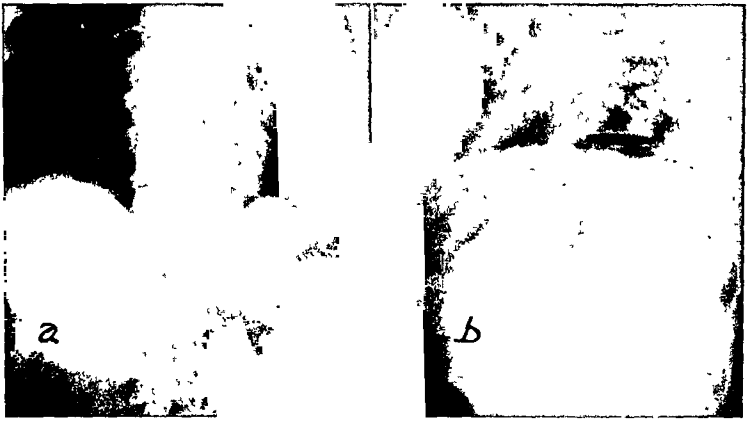


Fig. 2—*a*, Anteroposterior view showing blade in sagittal plane to the left of midline and at the level of the eleventh thoracic vertebra.

b, Lateral view of lower chest and abdomen showing knife blade through the eleventh thoracic vertebra and protruding anteriorly about an inch

Postoperatively the patient developed weakness of the right lower extremity and this gradually improved. The patient complained of moderately severe upper abdominal pain during the first one to two weeks of convalescence and had associated guarding and tenderness, probably on a reflex basis. Peristalsis was normal and the patient was able to eat without difficulty. On the seventh postoperative day, the patient exhibited a left Babinski reflex which indicated a beginning mass flexor withdrawal reflex and subsidence of spinal shock. This was significant prognostically since contusion and not laceration of the spinal cord had therefore occurred. The initial bladder catheter drainage was discontinued and the patient voided normally. He was discharged back to the prison on Dec. 12, Our case is unique, representing survival after successful closure of a perforating, transfixing stab wound of the descending thoracic aorta complicated by a Brown-Séquard syndrome due to concomitant spinal cord damage.

CASE REPORT

L. B., a 44-year-old prison inmate, entered the Hospital of the University of Pennsylvania, on Dec. 2, 1956, approximately twenty-seven hours after being stabbed in the back, chest, and left arm by a fellow prisoner. The previous medical history was not significant. The patient was given penicillin the day of injury and morphine for severe pain referred to the left hip and radiating over the distribution of the left sciatic nerve.

The physical examination revealed a well-developed, well-nourished, alert, 44-year-old Negro man in no acute distress. The vital signs were normal. There were multiple stab wounds of the left chest and arm, some of which were sutured. There was a puncture wound of the back about 2 cm. to the right of the midline at the level of the eleventh thoracic vertebra. The chest expanded equally and well and breath sounds were normal. The cardiac rhythm was regular and no murmurs were audible. The abdomen was soft and flat with normal peristalsis. Anal sphincter tone was good. All peripheral pulses were palpable and equal. The neurological examination revealed all cranial nerves intact, a flaccid paralysis of the left lower extremity from below and including the hip girdle muscles with intact sensation, loss of pain and temperature sensation over the right lower extremity below the gluteal fold, absent deep tendon reflexes in the left knee and ankle, and diminished abdominal reflexes on the left.

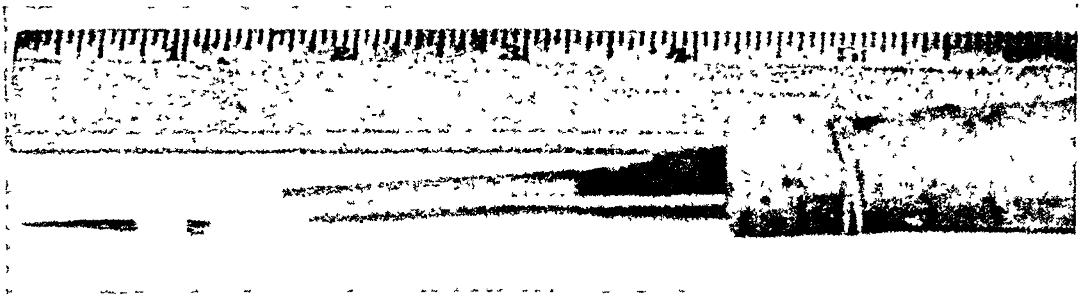


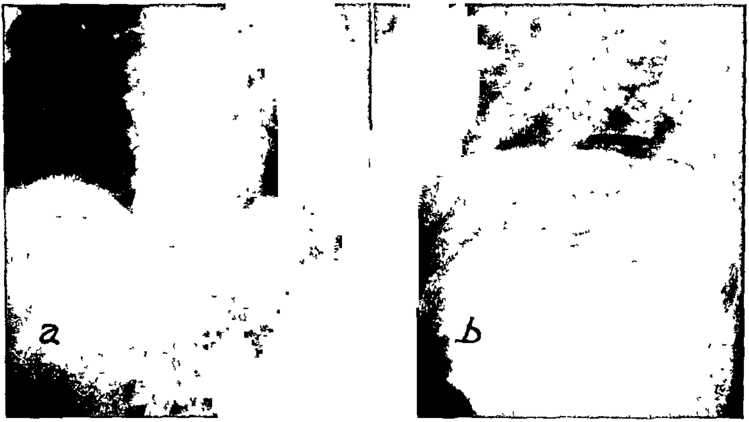
Fig. 1.—Stiletto and blade remnant with two pieces about one inch long removed from the anterior aspect of the eleventh thoracic vertebra.

The injuries were inflicted by a stiletto, ground from a steel file into a tapering, elliptical shape and affixed to a five-inch wooden handle (Fig. 1).

Studies revealed a hemoglobin of 16.5 Gm. and a leukocyte count of 12,700. Routine studies of blood chemistries were within normal limits. A lateral chest film showed a knife blade approximately 9 cm. long which had traversed the neural arch and body of the eleventh thoracic vertebra, the tip extending beyond the vertebral body and possibly transfixing the aorta. In the anteroposterior view, a metallic foreign body only 0.5 by 1.4 cm. was visible (Fig. 2). There was also a longitudinal fracture of the neural arch of the eleventh thoracic vertebra. No other abnormalities of the thoracic cage or contents were noted.

The patient was operated upon within two hours of admission. It seemed possible that the portion of the knife blade, protruding anterior to the body of the vertebra, transfixed the aorta. The remnant was to the left of midline and almost directly in a sagittal plane. It was elected to carry out a laminectomy over T_{10}, T_{11}, and part of T_{12} and, if the blade could be reached and removal deemed possible, thoracotomy would then be performed to safeguard against massive hemorrhage.

The patient was placed in the right lateral decubitus position and, under endotracheal anesthesia, a laminectomy over T_{10} and T_{11} was performed. Upon exposing the dura, no tear was seen. It was then opened and the spinal cord found to be slightly edematous and congested. A No. 14 rubber catheter was passed upward and downward along the cord for a distance of three or four inches without encountering a block. The knife blade could not be found. A thoracotomy was then carried out through the ninth left interspace. The diaphragm was partially opened to facilitate exposure. The point of the knife was then easily palpated within the thoracic aorta at the level of the eleventh thoracic vertebra about ⅛ of an inch from the anterior wall in the midline of the vessel. A retropleural hematoma was present around the aorta posteriorly from the diaphragm to about the ninth thoracic vertebra and consisted of approximately 200 c.c. of blood. The aorta was then mobilized, occluded above and below the point of entrance with Crafoord clamps, and lifted off the knife blade anteriorly. The laceration was 1.5 cm. long and was closed with a continuous suture of No. 4 0 arterial silk. The time of total occlusion of the aorta was seven minutes. Various attempts to manipulate the protruding knife blade were unsuccessful since it was firmly imbedded in the vertebral body. Finally it was broken off flush with the vertebral body and the remainder left in situ. The removed portions are shown in Fig. 1. The chest was closed with a large drainage tube connected to a water-seal bottle. The patient received a total of 4,500 c.c. of blood during these procedures which he tolerated very well. Postoperative x-rays revealed that the laminectomy was one vertebra too high, but even so it is believed that the blade remained extradural.



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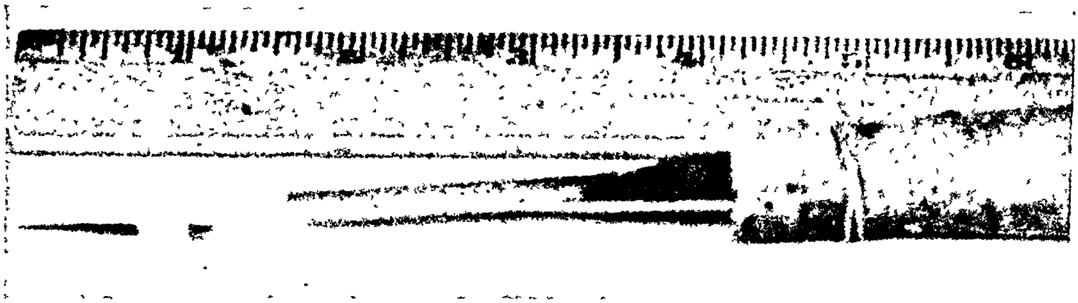

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BRONCHIAL CLOSURE WITH CATGUT SUTURES

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IN JULY of 1956, Bjork[1] published a report on suture materials used to close divided main and lobe bronchi. Approximately 29 of his 224 patients, with bronchial closure using nonabsorbable suture materials, developed suture granulomas, whereas none did so when the closure was effected with chromic catgut. His incidence of bronchopleural fistula did not increase when chromic catgut was used. Bjork also described the use of chromic catgut as early as 1929 by Brunn[2] for a one stage lobectomy.

Although the diagnosis of endobronchial suture granuloma per se was not generally used at Glenn Dale Hospital, the bronchoscopic findings when a bronchopleural fistula could be visualized implicated the suture as an undesirable foreign body in the infected bronchial stump. In surgery of other parts of the body, silk stitches as well as other permanent suture materials that have interstices have long been known to be undesirable foreign body foci. These can and will make surgical infections chronic and lead to sinus formation. In addition to the granuloma effect of a foreign body plus infection, the possibility of a foreign body promoting and/or potentiating a bronchopleural fistula is implied. It is important to us to have a dependable method of bronchial stump closure with absorbable suture material to avoid the foreign body effects of nonabsorbable material.

In addition to the role of silk sutures in infections, it is the opinion of the medical staff at the Glenn Dale Hospital that some patients have been known to suffer from a chronic cough which abates after the expectoration of a silk stitch. This was occasionally associated with a bronchitis similar to those reported by Pellnitz.[3] An exact tabulation of these cases of stitch cough is not possible except for the few that had stitches removed by the bronchoscopist. Nevertheless, the clinical experience of physicians following hundreds of postoperative cases for many years cannot be overlooked. This experience was continued even after the advent of drug therapy, although the number of such cases was thought to be considerably less since the beginning of streptomycin therapy.

To gain the advantages of absorption of suture material in bronchial stump closure, we began a clinical trial of chromic catgut closure of the bronchus in September, 1956.

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1956. A follow-up report two months later revealed that some atrophy of the left calf muscles had occurred but that considerable motor power had returned to the left lower extremity and pain sensation had returned to the right.

SUMMARY

The first known instance of successful closure of a stab wound of the extrapericardial thoracic aorta is reported. This represents the sixth known case of successful suturing of a thoracic aorta laceration. The descending thoracic aorta of a 44-year-old man was transfixed by a stiletto plunged through the body of the eleventh thoracic vertebra, contusing the spinal cord and producing a Brown-Séquard syndrome. The literature is briefly reviewed.

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were found to be smear or culture positive. In the cases of catgut closure, 21 patients had has similarly positive sputum smears or cultures preoperatively, or the tubercle bacilli were recovered from their resected specimens. The tubercle bacilli from sputa were found resistant to streptomycin, INH, or PAS in 5 cases of silk closure, and in 3 cases where the closure was effected with chromic catgut. All patients were bronchoscoped prior to resection. No patients with acute or subacute bronchial tuberculosis or bronchitis were resected. Histologic study of the bronchi at the resection line from the resected specimens was carried out and found to confirm the preoperative endoscopic impressions.

METHODS OF BRONCHIAL CLOSURE

A simple, single row of interrupted chromic catgut sutures was used to close the bronchi. The chromic catgut was open market purchase of Ethicon manufacture. In both the control and the catgut series the bronchi were transected and then closed with 3-0 silk or 2-0 chromic catgut on an Atraumatic needle. Occasionally, 4-0 chromic catgut was used when anatomically necessary. Each suture was placed approximately 5 mm. deep in the cut end of the bronchus. This type of stitch avoids the crowding of masses of suture material close to the cut tissues which tends to interfere with local blood supply. In addition, the use of excessive amounts of suture material would tend, in the cases of nonabsorbable silk, to enhance the possibility of granuloma formation. Undue tension on the suture line was avoided. An airtight seal was effected as evidenced by the usual under saline solution test, with increased endotracheal pressure provided by the anesthetist.

BRONCHIAL COMPLICATIONS

Table II summarizes the bronchial complications of each group. There were 5 postoperative pneumothorax problems in the control group of silk closure. All these occurred from the main, lobar, or segmental bronchi. Exact diagnoses as to these locations were accomplished by the clinical course, the x-ray picture and endoscopy in the 2 pneumonectomy cases, by bronchoscopy and x-ray studies in the one lobectomy and the one bilobectomy case, and by bronchogram in the one segmental resection case. In the catgut series, the fistula in the one pneumonectomy case was diagnosed by the clinical course, x-ray studies, and by the obvious result after re-intubation. The 2 cases of air leak fistula from the intersegmental plane in the catgut cases were diagnosed in one case by open thoracotomy and direct visualization at the time of thoracoplasty, and in the second case by the roentgenogram and the clinical course. This

TABLE II

	SILK	CATGUT
Bronchopleural fistula of main, lobe, or segmental bronchi	5	1
Air leak fistula of intersegmental planes	0	2
Total number of postresectional pneumothorax problems	5	3

METHODS AND MATERIALS

A total of 60 consecutive pulmonary resections for far advanced and moderately advanced tuberculosis in which all the bronchi were closed with chromic catgut were studied. As a control series, 60 consecutive pulmonary resections for surgically similar far advanced and moderately advanced tuberculosis in which all the bronchi were closed with silk stitches were compared with the chromic catgut cases. These control patients had had 60 similar types of pulmonary resections immediately preceding, in time, the catgut group. Two of the patients who had had silk closure had to be eliminated as they were surgically dissimilar by virtue of their having received intrathoracic injections of Varidase for hemothorax. Otherwise the consecutive order was rigidly followed. The methods of operation, postoperative care, and general management were not changed from group to group. For purposes of the study the pulmonary resections at the Glenn Dale Hospital were separated into 7 basic classes: pneumonectomy, 12 cases; bilobectomy, 5 cases; lobectomy, 15 cases; lobectomy together with simultaneous segmental resection, 7 cases; lobectomy with wedge resection, 6 cases; segmental resection alone, 9 cases; and finally, segmental resection together with wedge resection, 6 cases. The catgut closure cases were matched according to the type of procedure with consecutive silk closure cases as outlined in Table I, making a total of 120 patients studied.

TABLE I

	SILK	CATGUT
Total number of individual bronchi closed (main, lobe, or segment)	78	73
Total consecutive patients	60	60
Number of cases according to resection classes:		
Pneumonectomy	12	12
Bilobectomy	5	5
Lobectomy	15	15
Lobectomy and segmental	7	7
Lobectomy and wedge	6	6
Segmental resection	9	9
Segmental and wedge	6	6

In each case the actual number of bronchi sutured was tabulated. Only the main bronchus, the lobe bronchi, and the segmental bronchi were recorded. Very small bronchi, such as those in the intersegmental planes, were not involved in any special closure technique. The small difference of 5 bronchial closures between the groups is accounted for by the difference in bronchial anatomy on the right and the left sides, as well as whether or not the bilobectomy cases were done through the intermediate bronchus or as two separate lobes such as the upper and middle on the right.

All patients received vigorous antituberculous chemotherapy administered in this hospital until the lesions were considered to have reached maximum resolution by x-ray studies and clinical conference studies. In the silk closure cases there were 25 patients who had had a positive sputum for tubercle bacilli by smear or culture 6 months prior to operation, or the resected specimens drugs This, in general, is in agreement with Bjork's[1] findings (2) The number of bronchopleural fistulas of the bronchi closed with catgut was lower than the number of bronchopleural fistulas in the cases closed with silk The commonly known high frequency of bronchopleural fistula in resistant positive cases held true in the silk group in 4 cases out of 5, whereas 3 resistant positive cases in which catgut was used healed well The suture material was the only change in the method of treatment of these 2 consecutive groups of patients These data, together with the known effect of foreign bodies in infected areas, suggest that the absorbable material is more desirable than the silk material Further clinical study of the resistant positive cases in relation to use of absorbable suture material is indicated (3) In the years following resection the patients are likely to benefit from catgut closure because the possibility of sutures being coughed up or causing streaking or hemoptysis from the granuloma effect is unlikely when absorbable suture materials are used

SUMMARY

Closure of bronchial stumps by chromic catgut in pulmonary resections for tuberculosis has been found to be quite satisfactory and consistently good Sixty cases of 73 bronchi closed with catgut were compared with a control series of 60 surgically similar cases in which 78 bronchi were closed with silk Catgut closure appeared to be superior to silk closure The resistance of tubercle bacilli at time of surgery and the bronchial complications were discussed

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latter patient showed a slowly increasing pneumothorax immediately after original extubation of chest catheters that were placed in the hemithorax at the time of the operation. Re-intubation immediately resulted in full re-expansion and resolution of this pneumothorax. There was no fever or cough.

Resistance of tubercle bacilli to streptomycin, PAS, and INH, occurred in 5 of 25 control cases, in which tubercle bacilli were recovered from the sputum or the resected specimen. Of these resistant cases, 4 patients developed bronchopleural fistulas. In the chromic catgut series, there were 3 patients with similarly resistant tubercle bacilli, in a total of 21 cases in which tubercle bacilli were recovered from sputum or from the resected specimen. None of these 3 patients developed bronchopleural fistulas. In all the resistant cases, the patients were treated nevertheless with PAS, INH, and streptomycin, as well as the other new antituberculous chemotherapeutic agents such as viomycin and cycloserine, as such drugs become available. In 3 cases of resistant tubercle bacilli in which tuberculous bronchopleural fistulas developed, the bronchial stumps were visualized. In these instances, silk stitches were seen to be present and acting as centrally located foreign bodies surrounded by white and necrotic bronchial tissue. Removal of these stitches, when it was possible with the bronchoscope, seemed to aid the control of the tuberculous bronchitis. However, there was no dramatic cure of the tuberculosis in these patients with established tuberculous bronchitis when these stitches were removed. This study should not be construed to imply that such would be the case. Nevertheless the presence of these foreign bodies seemed deleterious and the pathogenesis of infection aggravated by foreign body was implied. One patient who had had segmental resection developed a nontuberculous silk stitch granuloma in a bronchial suture line, and was relieved of symptoms of cough and hemoptysis when the silk suture was removed and antibiotics were administered.

CONCLUSIONS

Sixty tuberculous patients had various types of pulmonary resections performed with 73 separate bronchi closed with chromic catgut. They had a satisfactory clinical course. Compared with the previous 60 surgically similar tuberculous patients who had 78 bronchi closed with silk, the 60 patients in the catgut series showed no increase of bronchial complications. In fact, there were 5 major bronchopleural fistulas in the silk group and only 1 major bronchopleural fistula in the catgut series. There were 5 resistant positive cases in the silk group, in which 4 tuberculous bronchopleural fistulas developed. There were 3 resistant positive cases in the catgut group and none developed bronchopleural fistulas. It is obviously impossible to follow the healing of bronchial stumps in patients by serial histologic studies. Further, it is recognized that the number of resistant cases is small and only possible trends can be recognized. Certain conclusions, however, have to be reached on the basis of clinical analysis and these are as follows: (1) Chromic catgut is as good as silk in effecting successful bronchial closure in resections for tuberculosis, whether or not the bacilli are resistant or sensitive to the usual antituberculous chemotherapeutic

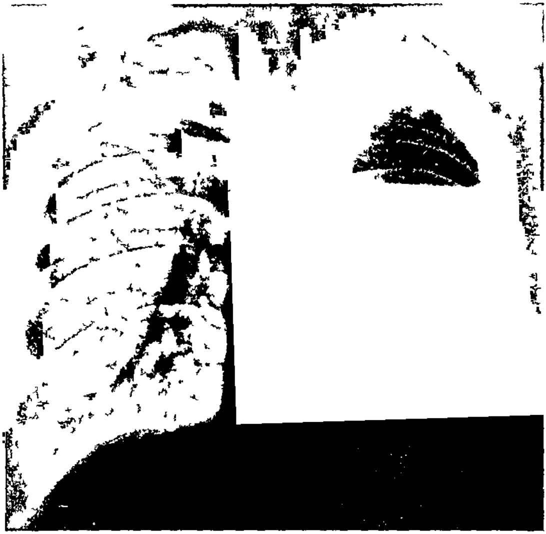

Fig 1 —Case 1 Roentgenogram showing complete atelectasis of the left lung with a massive effusion

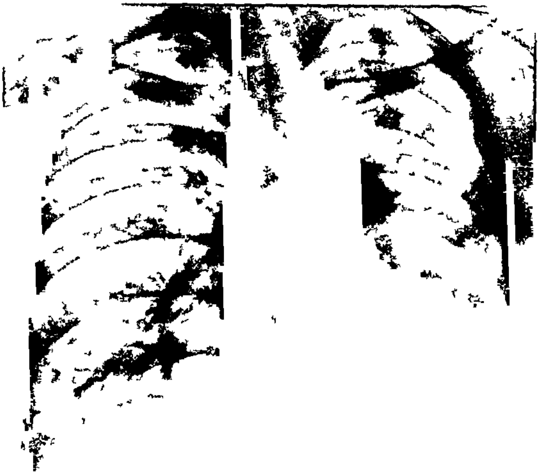

Fig 2 —Case 1 Roentgenogram made three months after operation shows good aeration of the left lung and a wide open left main bronchus

THE KINKED BRONCHUS SIMULATING A BRONCHIAL STENOSIS

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IN CASES of pulmonary tuberculosis with atelectasis of the lung and a tuberculous empyema or a fibrothorax, an extrapleural resection is usually performed. During the performance of such an extrapleural resection it is often easier to complete the operation if, during an early stage, the bronchus, the artery, and the veins are divided. This practice has only been followed in cases where the pulmonary tissue is obviously destroyed by multiple cavities. In all other cases, I strongly advocate that decortication be performed first in order to evaluate how much lung should be removed. Following this principle has made it possible to save functioning lung in many cases. In one instance the right lower and middle lobes could be saved after having been completely atelectatic under a tuberculous empyema for eleven years. One year later 33 per cent of the total oxygen uptake occurred on the right side in this patient now found negative by guinea pig test. Even patients coming to surgery under the diagnosis of a bronchial stenosis with atelectasis and fibrothorax should not be routinely treated by resection. The aim of this paper is to advocate a partial decortication with thorough examination of the bronchus before any hilar structure is divided. Following this principle it was possible to save pulmonary parenchyma in 2 cases with a preoperative diagnosis of bronchial stenosis. The stenosis was found to be due to kinking of the bronchus after dislocation and fixation of the lung by pleural effusion, and fibrous scarring.

CASE REPORTS

CASE 1.—A 54-year-old woman developed left-sided pulmonary tuberculosis in 1929. An artificial pneumothorax was instituted and continued until 1932. Her pulmonary tuberculosis was then considered healed and x-ray studies of the lung remained unchanged for twenty-four years. A high fever then occurred and new x-ray studies showed that the left lung was completely atelectatic with a massive pleural effusion (Fig. 1). Bronchoscopy showed a concentric stenosis of the left main bronchus, similar to an old tuberculous stricture, 3 cm. below the carina. Bronchography was performed to determine the possibility of bronchial resection with bronchotracheal anastomosis but a complete obliteration of the left main bronchus, 2 to 3 cm. below the carina was demonstrated. An extrapleural pneumonectomy was planned. At operation, a fluid-containing fibrotic pleural sac was found and liberated from the chest wall and diaphragm. A partial decortication was made and the lung found better than expected. The decortication was completed and now the lung could be filled with air under high pressure. Some small and hard nodules were palpated in the superior segment of the left lower lobe but there was no indication for resection. The left main bronchus was dissected free in its entire length and a longitudinal incision made. The stenosis, diagnosed by bronchoscopy and bronchography, could not be

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SUMMARY

In pulmonary tuberculosis, bronchial stenosis is a common finding. Of 151 pneumonectomies for tuberculosis, 37 cases (25 per cent) were performed for stenosis of the main bronchus and in 31 cases the stenosis was on the left side

In a series of 1,000 resections for pulmonary tuberculosis, 2 cases with a diagnosis of bronchial stenosis were found to have a kinked bronchus secondary to a dislocation of the lung due to pleural effusion and fibrosis. The cases illustrate the advantage of (a) decortication and inspection of the lung before division of the hilar structures, and (b) a longitudinal bronchotomy for verification or exclusion of a bronchial stenosis before the bronchus is divided in cases where the external appearance of the bronchus is not typical of a stenosis.

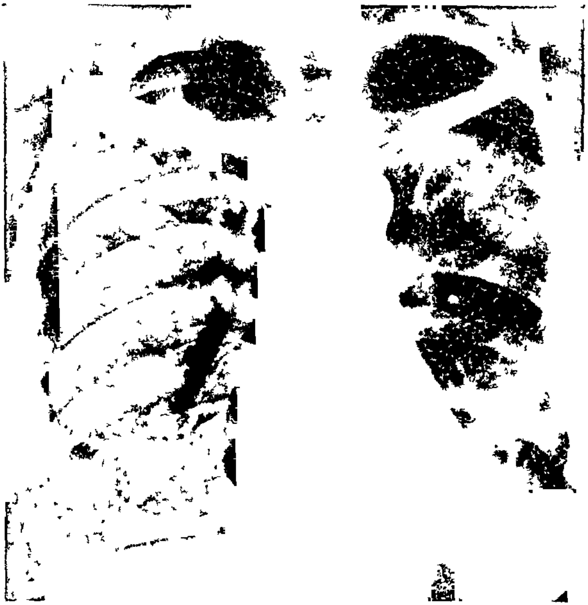

Fig 4—Case 2 Roentgenogram after decortication shows good aeration of the left lung

In 2 other cases a preoperatively diagnosed bronchial stenosis was, at operation, found to be due to kinking of the bronchus, but the lung was destroyed and had to be removed.

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found, but the mechanism of bronchial obstruction could be reconstructed. As a result of the dislocation of the lung, the left main bronchus was kinked. After liberation and decortication of the lung, the obstructing kink disappeared. The bronchial incision was closed by interrupted sutures of No. 3-0 Chromic catgut. The patient made an uneventful recovery apart from an atelectasis necessitating a bronchoscopy. Postoperative roentgenograms then showed good aeration of the left lung (Fig. 2).

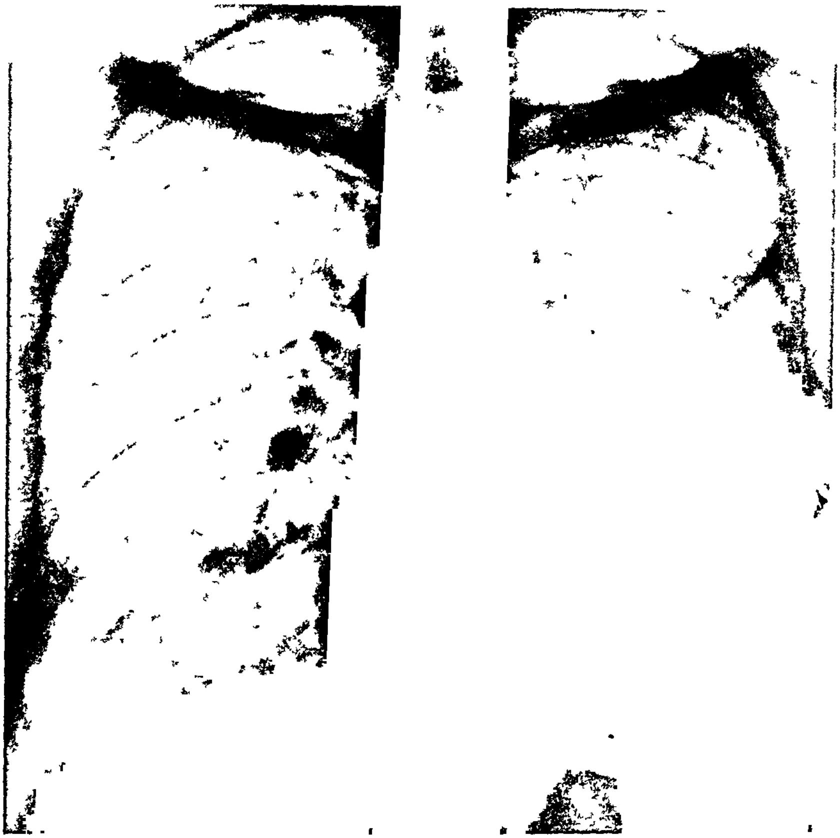

Fig 3.—Case 2. Roentgenogram showing left lower lobe atelectasis with intrapleural effusion.

Case 2.—A 58-year-old man had a left pleurisy in April, 1952. Guinea pig tests of sputum and pleural fluid were positive. Bronchoscopy showed a concentric stenosis of the left lower lobe bronchus. A tumor was suspected on two occasions, but biopsy was negative. On a preoperative diagnosis of left lower lobe atelectasis, due to a tuberculous bronchial stenosis or to a carcinoma (Fig. 3), an extrapleural lobectomy or pneumonectomy was undertaken. At operation (Oct. 24, 1952) the left lung was first completely freed by extrapleural dissection and decorticated. It was necessary to remove the visceral pleura over the lower lobe. After extirpation of the pleural sac, the left lower lobe was aerated. The left main and lower lobe bronchus was dissected free and found to be thin-walled and normal. As it was easy to palpate the normal wall of the lower lobe bronchus, having a 1 cm. internal diameter, this was not opened. There was no stenosis of the left lower lobe bronchus but the obstruction had been caused by kinking of the bronchus due to the fluid collection and fibrosis of the pleura. Immediately after the operation, bronchoscopy was performed and showed a normal left lower lobe bronchus. After removal of the tubes, an air pocket was formed which needed additional drainage for five days. Otherwise the recovery was uneventful and two months after the operation 32 per cent of the total oxygen uptake occurred in the left lung. In this case a planned lobectomy for bronchial stenosis ended up in a pleurectomy for a tuberculous pleurisy (Fig. 4).

tumor together with the infiltrated part of the bronchial wall should be carried out He also states that the scar of a bronchial suture line has a great tendency to shrink causing a stricture, and making bronchoscopic dilatation necessary

Goldman[4] reports the case of a 45-year old woman with a carcinoid adenomatous polypoid tumor of the bronchus which recurred 5 years after the entire tumor was thought to have been removed through a bronchoscope He found that it involved two rings of the right main stem bronchus just distal to the carina He had trouble controlling the proximal bronchus and used traction sutures on the carinal cartilage An encircling bronchial excision was made rather than a sleeve resection and the continuity of the bronchus was restored without loss of lung tissue

Gebauer[5] cites a remarkable case of Officer Brown of Melbourne in which the patient had an adenoma in the distal left main bronchus, opposite the upper lobe orifice, which obstructed the lower lobe bronchus and caused expiratory emphysema of the left lung The main stem bronchus was transected above the tumor, the upper lobe bronchus lateral to the tumor, the superior segment lower lobe bronchus posterior to the tumor and the basal bronchi transected below the tumor The distal upper lobe stump was then sutured to the anterior edge of the basal stump and the superior segment to the posterior edge Thus, the three divided bronchi were converted to a single stump which was anastomosed to the proximal stump of the main stem bronchus

These cases represent successful attempts at preserving the whole of the functioning lung by removing the local tumor along with varying portions of the underlining bronchial wall

We recently encountered a bronchial adenoma originating in the left mainstem bronchus which we believe is the first such case reported in which an entire sleeve of the main stem bronchus was excised with end to end restoration of the bronchus

CASE REPORT

L M, No 15755 56, a 19 year old white male had always enjoyed good health until 2 months prior to admission when he became aware of a short sporadic nonproductive cough This persisted and one month prior to admission (March 23, 1956) he became aware of the gradual onset of shortness of breath During this time he lost no weight, was not febrile, had no night sweats, no hemoptysis, and raised no sputum

Three days prior to admission he developed pain in the left posterior chest It was not pleuritic but aching in nature, and persisted to admission Just prior to admission he had two small episodes of hemoptysis A routine chest film in February, 1956, was interpreted as normal On questioning, it was learned that the patient was very fond of eating peanuts

Physical examination disclosed a temperature of 101° F, a pulse of 120, respirations of 32, a blood pressure of 132/50 mm Hg Examination of the chest showed definite lag in respiration on the left, diminished percussion note, diminished tactile and vocal fremitus, and diminished to absent breath sounds over the lower two thirds of the left chest Examination of the right lung was normal

A roentgenogram (Fig 1) disclosed an elevated left diaphragm with shift of the mediastinum to the left and some collapse of the left lower lobe The clinical impression was aspiration of foreign body (peanut) or bronchial adenoma Tomography did not reveal

BRONCHIAL ADENOMA TREATED BY SLEEVE RESECTION OF THE MAIN-STEM BRONCHUS

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ALTHOUGH bronchial adenomas usually do not metastasize, they are locally invasive and must be considered potentially malignant tumors to be treated by wide local excision. When they involve the main-stem bronchus, adequate wide excision has generally meant pneumonectomy. In recent years, however, a number of cases have been reported in which the employment of bronchial anastomosis has resulted in the cure of main-stem bronchial adenomas by lobectomy rather than pneumonectomy. Only a few cases of local excision of the tumor and of portions of the bronchial wall with reconstruction of the bronchial tree without loss of lung tissue have been reported.

D'Abreu and MacHale[1] report the case of a 48-year-old woman with an adenoma of the left main-stem bronchus just below the opening of the left upper lobe bronchus which was treated by excising the entire thickness of the bronchial wall containing the tumor, leaving a minute strip on the anteromedial wall. The repair was by an end-to-end anastomosis using linen thread sutures. A follow-up bronchoscopy 25 months after operation showed the lumen at the anastomotic site to be two thirds of normal size.

Crafoord[2] cites the case of a young man in whom an adenoma infiltrated a part of the bronchial wall from just above the right upper lobe bronchus down to below the middle lobe bronchus. At thoracotomy, it was possible to do a radical local excision of the extrabronchial plum-sized part of the tumor, the infiltrated part of the bronchial wall, and the intrabronchial tumor with subsequent plastic repair of the bronchus. There was a tendency to stricture formation at the site of the bronchial repair which has made bronchoscopic dilatation necessary. Crafoord states that Price Thomas had a case which he successfully treated in the same manner.

Björk,[3] from Crafoord's clinic, states that in most cases of mucous and salivary gland tumors (adenoma and cylindroma) it is still necessary to perform a lobectomy due to long-standing bronchial obstruction causing bronchiectasis. But in early cases, when the changes behind the tumor are reversible a lung-sparing course is followed. He recommends that most of the intrabronchial portion of the tumor be electrocoagulated, giving a free air passage with re-expansion of the atelectasis and drainage of accumulated secretions. Some weeks later a block removal of the intra- and extrabronchial part of the

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Before the bronchus was transected it was realized that some provision had to be made to prevent the proximal bronchus and carina from retracting into the mediastinum out of the operative field. Retention sutures would probably not hold the marked tension necessary to bring the carina into view and, in addition, would have been in our way. Therefore, by blunt dissection with the fingers, the trachea just above the carina was freed and, after an intratracheal catheter was passed beyond the carina into the right main-stem bronchus, a tape was passed around the trachea and tension was placed on it so the proximal edge of our resection was brought well into the operative field (Fig. 2). A complete sleeve of the main-stem bronchus comprising 3 cm. from just below the carina to just proximal to the left upper lobe take-off was excised and without tension the two ends were brought together using interrupted 3-0 silk sutures. Following this reconstruction the lung expanded and collapsed quite well and the suture line retracted into the mediastinum. Although the fate of the lung was still uncertain, it seemed that it had a reasonable chance of surviving. The chest was closed using two chest tubes for suction drainage.

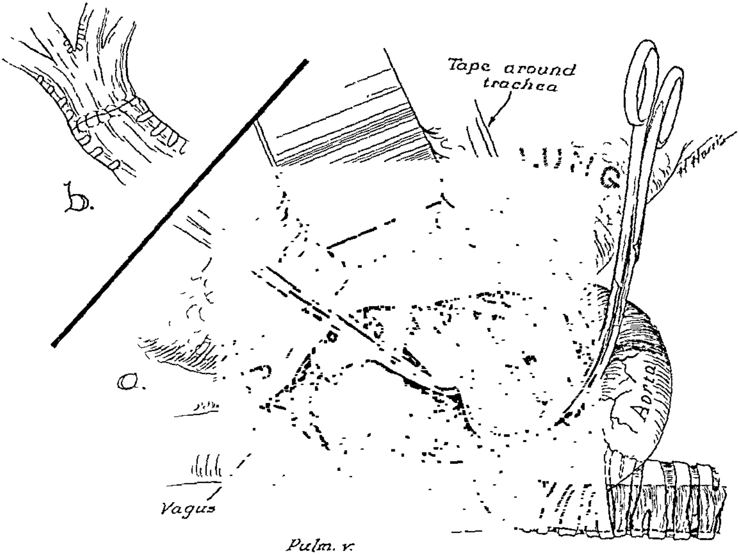

Fig. 2.—*a*, Operative field showing adenoma in left main-stem bronchus. *b*, Completed end-to-end anastomosis after sleeve resection of the bronchus.

Immediately following the operation, the patient's temperature returned to normal from the preoperative level of 101° F. The patient's postoperative course was completely uneventful. Roentgenograms of the chest showed that the mediastinal shift to the left returned to the midline and the left diaphragm again functioned normally (*Fig.* 3). The creamy material that extruded from the lung after bronchotomy was sterile on culture. Bronchoscopy, three weeks after operation, disclosed a patent left main-stem bronchus with some irregularity of the anastomotic site. The silk sutures could be visualized. Bronchoscopy, 8 months after operation, disclosed a completely full-sized left main-stem opening with no sutures or other evidence of a visible anastomosis. The bronchoscope was [illegible] any bronchial pathology. Bronchoscopy was performed three times. On the first two occasions massive bleeding was encountered as soon as the bronchoscope was introduced into the left main-stem bronchus and the examination had to be abandoned. On the third attempt a whitish friable tumor was seen about 2 cm. beyond the carina. Biopsy revealed adenoma of the bronchus. The tumor appeared to fill the entire lumen of the bronchus. From the anatomic location of the growth it was felt that a pneumonectomy would probably be necessary to provide an adequate resection.

Operation.—On April 27, 1956, under general intratracheal anesthesia, the left chest was opened through a posterolateral incision resecting the fifth rib and dividing the fourth and sixth ribs posteriorly for more adequate exposure. At operation, no collapse of the left lung was noted, but rather it was distended and firm and could not be compressed by manual pressure. It was obvious that a main-stem bronchial obstruction was present. Palpation of the entire lung and its hilum did not reveal any masses. The left main-stem bronchus was freed from the carina to the left upper lobe takeoff and, by palpating the bronchus, an olive-sized mass could be felt through its wall just beyond the carina in the main-stem bronchus. It could not be visualized from the outside of the bronchus. It was elected to open the bronchus to delineate the pathology and visualize the problem. A 3 cm. longitudinal opening was made through the lateral wall of the main-stem bronchus. At first the thought of a local excision across the pedicle with cauterization of the base was considered but the base of the tumor was relatively extensive and such a course could not be defined as an adequate local excision. It was obvious that a generous local excision would necessitate the removal of a complete sleeve of at least 3 cm. of the left main-stem bronchus.

Fig. 1.—Admission film showing shift of mediastinum to left with interpretation of atelectasis of left lower lobe.

It should be mentioned here that, when the bronchotomy was first made, enormous quantities of thick white mucoid material extruded from the distal bronchus. The lung was then completely decompressed by squeezing it with extrusion of the above described material from the distal cut bronchus. Culture was taken of this material. Although its appearance and the clinical febrile course of the patient suggested massive infection of the left lung, it was elected to try to save the lung realizing that if the lung was irreversibly damaged a pneumonectomy might be necessary later on.

of the bronchus and lung, this case demonstrates what can be done with bronchoplastic techniques and a plea is made to explore such a course before resorting to the usual pneumonectomy in these cases

SUMMARY

1 Bronchial adenomas are potentially malignant tumors and must be treated by wide local resection

2 When adenomas occur in the main stem bronchus, pneumonectomy has been the procedure of choice

3 A case of an adenoma lying within and completely obstructing the left main stem bronchus in a 19 year old male is presented

4 A wide local resection was performed by removing a 3 cm complete sleeve of the bronchus with end to end anastomosis of the bronchus and no sacrifice of lung tissue

5 The anastomosis was made possible technically by mobilization and traction of the trachea above the carina

6 Although the left main stem bronchus had apparently been totally blocked for several months, the retained secretions were sterile and no irreversible damage of the lung could be demonstrated

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passed through the area of the anastomosis into both the left upper and left lower lobes of the lung. A postoperative bronchogram revealed a normal appearing left bronchial tree without evidence of bronchiectasis.

Comment: This case represents one which, we had expected would require a pneumonectomy but was successfully treated by a sleeve resection of the main-stem bronchus.

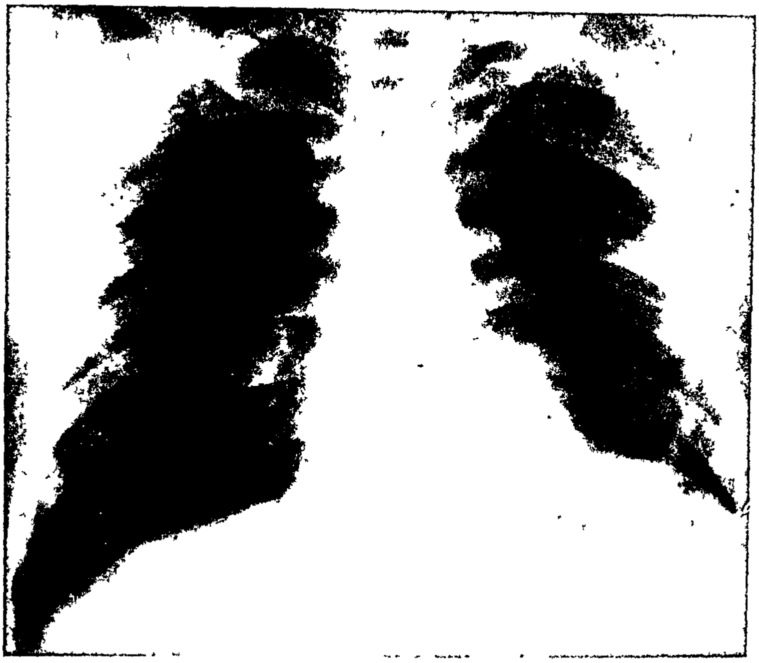

Fig. 3.—Postoperative film showing clear left lung field and mediastinum returned to the midline.

DISCUSSION

We believe this case represents the first complete sleeve resection of a main-stem bronchus with successful reconstruction for adenoma of the bronchus. This is not technically difficult if the trachea above the carina is mobilized and pulled downward by an encircling tape. The problem of postoperative stenosis or stricture formation is insignificant if a complete sleeve of bronchus is removed rather than leaving a bridge of tissue on one wall. Where a sleeve is taken the anastomosis is even and symmetrical without the puckering or distortion that results in stenosis or stricture formation. This patient's symptoms and admission x-ray studies suggest that the left main-stem bronchus was blocked completely for from 2 to 3 months, yet the cultures were sterile at operation and no bronchiectasis was found by bronchogram in the blocked lung. Thus, it appears that fairly long-standing complete bronchial obstruction does not necessarily produce any irreversible damage to the lung, providing infection is not present. Such local resections, of course, predicate an adequate margin of resection lest the tumor recur at the anastomotic site as these neoplasms are prone to do if completely removed.

Although a completely intraluminal bronchial adenoma lying within the main-stem bronchus must be a relatively rare anatomic finding in adenomas

TABLE I

	AUTHOR	YEAR	AGE	SITE	GYNECOMASTIA	PREGNANCY TEST
1	Askanazy[15]	1906	19	Hypophysis	--	--
2	Miller and Browne[16]	1922	39	Retroperitoneal	--	--
3	Arendt[17]	1931	20	Anterior mediastinum	Yes	-
4	Kantrowitz[18]	1932	22	Mediastinum teratoma, chorioepithelioma, metastasis	No	Pos
5	Fenster[19]	1934	27	Retroperitoneal	Yes	Pos
6	Gerber[20]	1935	23	Retroperitoneal	No	Pos
7	Weinberg[21]	1939	70	Bladder	No	Pos
8	Mathieu and Robertson[22]	1939	27	Retroperitoneal	Yes	Not done
9	Ferrarini[23]	1940	20	Retroperitoneal	No	Pos
10	Erdmann, Brown and Shaw[24]	1940	20	Retroperitoneal	No	Pos
11	Hyman and Leiter[25]	1943	57	Bladder	--	Pos
12	Plenge[26]	1944	30	Retroperitoneal	--	--
13	Stowell, Sachs, and Russell[10]	1945	15	Diencephalon	No	Pos
14	Laipply and Shipley[3]	1945	13	Mediastinal	Yes	Pos
15	Hirsch Robbins, and Houghton[8]	1946	26	Mediastinal	Yes	Pos
16	Ferreira Berrutti[27]	1946	37	Lung and liver, no definite primary	No	
17	Giroux and Desmeules[28]	1947	12	Mediastinal	No	Pos
18	Dvoracek[29]	1949	24	Prostate	Yes	Pos
19	Shlimovitz and Van Brown[5]	1950	27	Mediastinal	Yes	Pos
20	Zorzi and Piacenti[30]	1952	29	Mediastinal	Yes	Pos
21	Nielubszyc[31]	1952	38	Mediastinal	Yes	Pos
22	Lynch and Blewett[32]	1953	26	Mediastinal	Yes	*
23	Pasquali[33]	1954	30	Retroperitoneal	Yes	Pos
24	Lochmann[34]	1954	61	Liver	Yes	-

*Not mentioned in original case report

There were no other children His mother was living and well His father was living six months post hemicolectomy for adenocarcinoma His mother had had a dermoid tumor removed from the right ovary soon after the patient's birth The tumor was diagnosed at the time she was pregnant

Physical examination revealed a well developed, obese white male, weighing 240 pounds Blood pressure 140/80 mm Hg, pulse 74, respiration 20 There was hypertrophy of both breasts, but because of his weight this was difficult to classify as gynecomastia A port wine nevus was present over the right arm and shoulder Healed inguinal herniorrhaphy and appendectomy incisions were present The genitalia were normal The physical examination was otherwise noncontributory

Laboratory data Hemoglobin 14 7, hematocrit 46, W B C 9,100 with a normal differential, urine, normal, total blood proteins 7 5, A G ratio 1 08, albumin 3 92, globulin 3 6, Kunkel globulin 16 5, V D R L (serologic test for syphilis) negative, electrocardiogram normal

X ray showed an anterior superior mediastinal tumor Skull plates and barium swallow were negative

Hospital Course—The patient had a right thoracotomy performed on Sept 7, 1956, and a large globular irregular encapsulated mass was removed from the anterior superior mediastinum The tumor was well encapsulated and there was no evidence of regional node involvement

When the microscopic diagnosis was returned, showing areas of choriocarcinoma, a urine specimen was obtained for gonadotropin assay On the fourth postoperative day this was quantitatively positive to 0 1 c c dilution The patient's postoperative cours

PRIMARY EXTRAGENITAL CHORIOEPITHELIOMA IN THE MALE MEDIASTINUM

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PRIMARY extragenital, mediastinal chorioepithelioma, although uncommon, is strangely enough a disease peculiar to men. The first proved case in a female was reported in 1952.[1] Although first described in the male mediastinum by Ritchie,[32] in 1903, mediastinal chorioepithelioma usually represents metastasis from a testicular cancer. Most authors maintain that every case must have a meticulous serial block section of the testes before an extragenital primary chorioepithelioma can be diagnosed,[2-5] and that any case showing small cysts or scars in the testes must be discarded, since they may represent primary testicular tumors undergoing involution.[4, 6, 7, 11]

Others require less stringent criteria, believing careful gross examination and representative sectioning sufficient.[8, 9] It is reasoned that small cysts or scars do not necessarily represent a tumor, but may represent the reciprocal effect of the extragenital hormone production. It is likewise pointed out in relation to mediastinal primaries, that testicular tumors seldom show anterior mediastinal deposits. In one series of 220 cases of metastasizing testicular tumors, none showed anterior mediastinal spread.[8] Some authors[10] question the validity of spontaneous healing of any testicular tumor. Thus, the valid number of reported cases of extragenital chorioepithelioma in the male, though small, will vary with criteria. This is further confused by the microscopic picture, terminology, and origin of extragenital chorioepithelioma in the male.

Up to 1957, 24 accepted cases of extragenital chorioepithelioma in a male have been reported in the literature (Table I). This represents the twenty-fifth case which has been proved by autopsy and serial section of the testes, excluding those cases which showed evidence of testicular scars or cysts, and is the tenth case of mediastinal origin.

CASE REPORT

D. B., a 20-year-old white male college student, was admitted to The George Washington University Hospital on Sept. 4, 1956, because of a radiopaque shadow in the right hilar region found for the first time on routine x-ray two months prior to admission. The patient was asymptomatic except for occasional left posterior chest wall pain radiating anteriorly in the month prior to admission, and had no history of pulmonary disease, weight loss or anorexia.

The patient had had a right inguinal herniorrhaphy at one year of age, a left inguinal herniorrhaphy at age 6, and an appendectomy at age 7. The past history and review of systems were, otherwise, noncontributory. A chest roentgenogram taken in January, 1956, was negative.

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Most authors believe that extragenital chorioepithelioma arises from a multipotent or totipotent cell that has become separated at the morula or blastula stage, or from germinal cells in the urogenital ridge which in the 8 mm embryo extends from the sixth cervical to the third lumbar region. The former could explain the bizarre extragenital locations reported, including the stomach, liver, and kidney. The latter explains the primarily retropleural or retroperitoneal location.

Since germinal extragenital tumors are by no means rare, it is surprising that so few with chorioepitheliomatous elements have been reported. This may be explained by the natural tendency for a short life span of placental tissue.

Some authors, however, doubt if any case is truly extragenital.[12] They also site the length of the urogenital ridge and the fact that ectopic testicular tissue has been found in the adult along its extent, in addition to the normally located testicular tissue. The previously held theory of mediastinal germinal tumors being of thymic origin has also recently come under criticism. It has been shown that thymic carcinoma can microscopically strongly resemble germinal tumors, primarily seminomas and, in many instances, has been classified as such.[13]

If we accept the concept of development as diagrammed above, the finding of extragenital chorioepithelioma does not represent as dramatic a cellular aberration as previously considered. As most cases reported were in reality germinal tumors with various elements in addition to chorioepithelioma, it is questionable if such a case should ever be reported as an "extragenital chorioepithelioma." However, since such tumors do represent the trophoblastic phase of a germinal growth rather than the more common embryonal or somatic development, some justification for this terminology may still be present, particularly since so relatively few cases of trophoblastic response have been seen in male extragenital germinal tumors. Whether or not this is true will depend on how diligently chorioepithelioma is sought in other primary extragenital germinal tumors. If so, extragenital chorioepithelioma may be relatively common, and classifying it as a distinct entity unjustified.

The clinical response to a malignant germinal tumor will vary with the cellular predominance. If chorioepithelioma is appreciable, the hormonal and secondary sex changes are apparent and the survival time diminished. This element with its inherent invasive ability leads to widespread metastasis and rapid death. The elaboration of chorionic gonadotropin, which is specific to the chorionic villi, is one of the characteristic diagnostic points. It produces the positive urine or serologic pregnancy test, and also gynecomastia, the latter having been experimentally reproduced in men, by Klatsen and Munson,[14] by daily administration of chorionic gonadotropin.

Most cases of extragenital chorioepithelioma develop varying degrees of testicular atrophy. These authors have also demonstrated the role of chorionic gonadotropin in the complex hormonal imbalance leading to this. Although frequently suggested as a means of determining postoperative residual tumor in resected cases, quantitative serial determinations of gonadotropin have not proved reliable as prognostic criteria. As illustrated in the above case, somatic complicated by pain in the left hip region. Roentgenograms suggested metastatic involvement of the left ilium, but no definite diagnosis could be made. He was discharged home on the eighth postoperative day.

Pathology Report.—The specimen was a single mass of tissue, lobular in shape, measuring 11.5 cm. by 9 cm. by 6.5 cm. The external surface was smooth, glistening and varied in color from light yellow to light red. An occasional irregular dark red hemorrhage was noted beneath the capsule. In some areas on the external surface, there was a suggestion of slight or gradual lobulation. The specimen had a soft, rubbery, consistency. The cut surface was soft, orange, and friable. It was focally necrotic and focally hemorrhagic. The periphery was more solid, had a yellow color and was nodular. The central softer necrotic areas appeared laminated and had the consistency of fibrinous material.

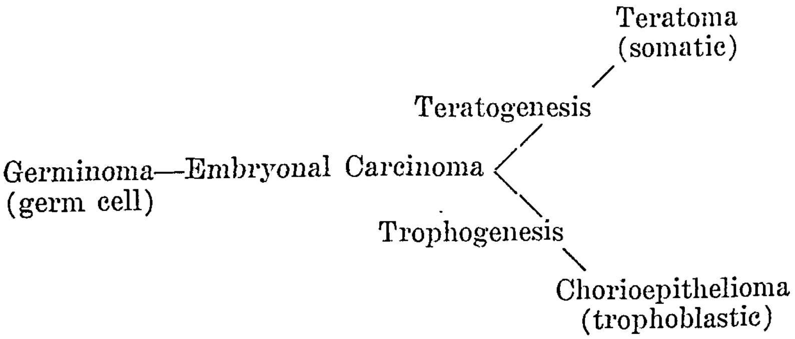
Microscopic diagnosis: Germinal tumor. The prime elements in the extragenital germinal tumor were seminoma and embryonal carcinoma, with areas of choriocarcinoma. The tumor appeared to have been completely removed, but vascular invasion within this tumor and the clinical course of similar cases made the prognosis uncertain. Thymic tissue was not identified.

The remainder of the patient's clinical course was rapidly downhill because of extensive skeletal and retroperitoneal metastasis. Deep x-ray therapy was administered at another institution, but he continued his downhill course, developing a cord compression paralysis which required a thoracic laminectomy from T_6 to T_8 on Oct. 9, 1957. The patient was re-admitted to The George Washington University Hospital on Jan. 18, 1957, in a comatose state, showing evidence of widespread metastasis, and he died Jan. 21, 1957.

Autopsy Findings.—Germinal tumor composed of seminoma, embryonal carcinoma, and chorioepithelioma, primary in the anterior mediastinum and surgically removed, with metastasis of the embryonal element to lungs, liver, bladder, esophagus, vertebrae, calvarium, left ilium, ribs, and brain. Bronchopneumonia, bilateral. Serial sectioning of both testes was negative for germinal tumor, but did show azospermia. Gynecomastia was not present.

DISCUSSION

The term extragenital chorioepithelioma is misleading. It is doubtful if any case actually represents a pure extragenital chorioepithelioma. Most cases represent chorioepitheliomatous elements in a germinal tumor, for the most part a teratocarcinoma. Recent studies have produced greater elucidation of the problem of teratogenesis. Most authors have conceded that gonadal teratoid growth originate from a totipotent germinal element of some sort, but extragenital teratomas have almost invariably been considered the result of aberrations in somatic development. How chorioepithelioma, which represents the trophoblastic or placental element, could be somatic in origin has always been difficult to assess. Recently, Friedman[11] has shown that extragenital germinal tumors are histologically identical to testicular tumors and, has, by comparative morphogenesis, set forth the following germinal origin and development, for both sites.



The hormonal elaboration, age, and the development of gynecomastia have been briefly discussed and stressed as diagnostic criteria in these cases. Treatment consists of radical excision.

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tumor elements may continue to progress despite a falling or negative quantitative gonadotropin determination. Since embryoma, seminoma, and teratoma have also been shown to give a positive urine or serum pregnancy test, this in reality may represent unidentified chorioepitheliomatous elements, since the responsible hormone is supposedly specific to the chorionic villi.

Radiologically, there is nothing characteristic of chorioepithelioma in the differential diagnosis of retroperitoneal or mediastinal tumors. However, if the possibility of chorioepithelioma is considered, the diagnosis can be made by a urine or serum pregnancy test in most cases before a tissue diagnosis is obtained. Although other germinal tumors do give a positive qualitative chorionic gonadotropin determination, chorioepithelioma gives a highly positive quantitative determination and in this manner can be differentiated from other germinal elements. Gynecomastia has been found to be present in over 50 per cent of the recorded cases. This may be unilateral in varying degrees of development, so that careful inspection is necessary. Although this tumor may occur at any age, as seen in the accompanying table, the greater number occur in the young adult. Since these tumors are from germinal anlage, and therefore dormant for many years, one may speculate that the hormonal effect of puberty gives some stimulus to the trophoblastic development of the germinal cells. It is interesting in this respect that this patient's mother developed a dermoid tumor of the ovary when pregnant with him, which was subsequently removed after his birth. An hereditary tendency for the development of germinal tumors in this family is suggested.

The treatment of chorioepithelioma is extremely unsatisfactory. Most patients die rapidly of metastasis. Lochmann[31] mentions several cases reported in the European literature of treatment with transfusions of blood from pregnant women. The theory is that even with a normal pregnancy, cells from chorionic villi invade the maternal vascular system stimulating an antibody response and a passive immune protection. Similar cases have not been reported in the American literature. Since chorioepithelioma is highly radioresistant, early diagnosis is imperative and radical excision the only treatment.

SUMMARY

This is believed to be the twenty-fifth reported case of primary extragenital chorioepithelioma in a male, proved by autopsy and serial section of both testicles. It is the tenth such case of mediastinal origin. We have reviewed the criteria for such a diagnosis and have listed the generally accepted cases.

The origin and morphogenesis of germinal tumors have been reviewed and while extragenital primaries have been in the past considered to be of somatic origin, recent workers have shown by comparative morphogenesis the germinal cell development of extragenital primaries as well as of genital primaries. We have pointed out that while most cases are reported as "extragenital chorioepithelioma," they are in reality germinal tumors in which chorioepitheliomatous elements have developed and it is questionable if these cases should be reported as a distinct entity.

incidence in our series (35 per cent) with the incidence reported in a recent comprehensive review[1] of mediastinal tumors in general (1.5 per cent). Considering that the latter figure was based on an analysis of 855 cases collected not from individual case reports but from all previously reported series, in other words an unselected group, one might conclude that the smaller figure provides a much more reliable estimate than does our own of the relative incidence of the mediastinal granuloma. This is supported by the rather meager literature on the subject, most of which has been devoted to the rarity and bacteriologic inscrutability of these tumors. Judging from our own series, even their granulomatous nature is liable to be missed. Although conceding that our experience is too limited to permit any emphatic conclusions, we suspect nonetheless that more careful study will establish both the true prevalence and, in many instances, the specific cause of the mediastinal granuloma. Such has proved true in our own investigations.

Grace[2] (1942) is credited with being the first to report the surgical excision of a so-called tuberculoma of the mediastinum. In 1944, the rarity of the lesion and its presumably tuberculous origin were further attested by a report by Blades and Dugan[3] of a posterior mediastinal tuberculoma in which only a very few acid-fast bacilli could be demonstrated. In 1950, Samson and co-workers[4] presented 4 more resected "tuberculomas" but raised some doubt about their exact etiology. Apparently the first ones to suggest that this might be a relatively common lesion were Storey and Lyons[5] who encountered 6 cases in a short period of time. Further doubt as to the cause of such granulomas was expressed by the Mayo Clinic group in 1954.[6] To the 16 cases culled from the literature they added another 16 cases in none of which were they able to establish a specific cause. For this reason they championed use of the term granuloma rather than tuberculoma when referring to such lesions. Subsequent reviews[7, 8] have re-emphasized the surgical aspect of these tumors especially when symptomatic or when a more sinister tumor cannot be ruled out.

As far as we can determine there was never any attempt to incriminate histoplasmosis as a cause until Samson did so in 1955,[9] and in that particular case his conclusion was based purely upon strongly positive histoplasmin and negative tuberculin skin tests. That same year Puckett[10] and, later, Schwarz[11] were able to demonstrate Histoplasma in resected mediastinal lymph nodes. Moreover, from analogous granulomatous lymphoid masses in the pulmonary hilus *H. capsulatum* has been cultured[12] and seen in specially stained tissue sections.[13, 14] More recently, Gillespie[15] has reported a case in which a mediastinal granuloma producing superior vena caval obstruction was attributed to histoplasmosis because of a positive complement-fixation test.

An analysis of our own series of mediastinal granulomas is presented in Table II. The patients were all young adults whose ages varied from 19 to 36 years. There were 7 men and 5 women; 10 white patients and 2 Negroes. Noteworthy features in this group include (1) a very low percentage of correct preoperative diagnoses, (2) uniformly negative bacteriologic yield in those cases in which the granulomatous contents were cultured, (3) the frequency with which the pathologist mistook the lesion for some totally different tumor such as a cystic teratoma or bronchogenic cyst, (4) the remarkably high incidence of

MEDIASTINAL GRANULOMAS

A Revised Concept of Their Incidence and Etiology

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THIS paper, while dealing with a representative group of mediastinal tumors, is concerned not so much with the mere documentation of another series but more specifically with investigation of the true incidence and etiology of what is generally regarded as an insignificant segment of mediastinal neoplasia, namely, the granuloma.

Generally recognized is the fact that tumors of the mediastinum, although affording the roentgenologist a fascinating diagnostic exercise, almost invariably defy a precise preoperative diagnosis. Less widely appreciated, however, are the problems one may encounter in identifying and classifying any given tumor even with the specimen in hand and histologic sections analyzed. This is graphically demonstrated by the present series in which the gross pathologic findings were commonly deceptive and microscopic interpretation was too often nonspecific. Only after painstaking study has the etiology of some of these tumors been clarified.

As shown in Table I there have been thirty-seven operations for primary mediastinal tumors at this hospital in the past decade. On the whole this series is comprised of a fairly representative group of mediastinal tumors with the notable exception of an inordinately high number of granulomas. That this is an extraordinary reversal of the commonly accepted incidence of granulomas relative to other mediastinal tumors is best shown by comparing the

Table I. Thirty-Seven Primary Mediastinal Tumors

TUMOR TYPE	BENIGN	MALIGNANT
Thyroid	2	–
Thymoma	2	2
Teratoma	–	3
Pericardial cyst	5	–
Lipoma	1	–
Neurogenic	4	2
Foregut cyst		
Bronchogenic	3	–
Esophageal	1	–
Granuloma	12	–
Totals	30	7

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The opinions or assertions contained herein are the private ones of the writers and are not to be construed as official or reflecting the views of the Navy Department or the naval service at large.

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II

GROSS APPEARANCE	SURGICAL PROCEDURE	SPECIAL FINDINGS	INITIAL PATHOLOGIC DIAGNOSIS	FINAL DIAGNOSIS
Thick walled cyst 6 cm in diameter filled with "sebaceous material" adherent to trachea, superior vena cava, and azygos vein	Resection	--	"Cholesteatoma"	Granuloma
Dense fibrous mass 6 × 4 cm in diam adherent to trachea superior vena cava and azygos vein	Biopsied		"Cicatrix"	Granuloma
Thick walled cystic tumor 5 cm in diam adherent to trachea, superior vena cava and filled with thick white material	Resection	--	Bronchogenic cyst	Histoplasmic granuloma
Dense matted mass of fibrous tissue adherent to esophagus	Biopsied	--	Sclerosing mediastinitis	Granuloma (sclerosing mediastinitis)
Cystic tumor 5 cm in diameter adherent to trachea, caseous content	Resection	-	Tuberculoma	Histoplasmic granuloma
Solid homogeneous mass 5 × 4 × 3 cm with reddish brown granular cut surface	Resection	Normal angiocardiogram	Infectious mononucleosis	Granuloma
Mass of matted lymph nodes measuring 8 cm in diameter, middle lobe studded with subpleural nodules	Biopsied	--	Tuberculoma	Histoplasmic granuloma
5 × 3 × 3 cm mass of lymph nodes	Resection	--	Boeck's sarcoid	Boeck's sarcoid
Large mass of matted lymph nodes	Biopsied	Refractile crystals seen with polarized light	Silicotic granuloma	Silicotic granuloma
Multiloculated thick walled cystic mass 4 cm in diam with shaggy inner wall and thick pasty sebaceous contents	Resection	--	Cystic teratoma	Granuloma
10 × 5 cm bilobulated cystic mass containing thick white creamy material, adjacent margin of rt lower lobe presented a subpleural nodule	Resection	"Rare acid fast bacillus"	Tuberculoma	Histoplasmic granuloma
4 cm mass of lymph nodes with thick white creamy contents, adherent to vagus nerve	Resection	--	Tuberculoma	Histoplasmic granuloma

TABLE

PATIENT	AGE	SEX	RACE	SYMPTOMS	SKIN TESTS			LOCATION	PREOPER. DIAGNOSIS
					TBC	HISTO	COCCI		
No. 1 (N. G. M.)	36	F	W	Cough of 2 months' duration	Not recorded			Rt. paratracheal	Lymphoma; bronchogenic cyst
No. 2 (J. L.)	26	F	W	Mild superior vena cava syndrome	Not recorded			Rt. paratracheal	Lymphoma
No. 3 J. E. H.)	27	F	W	*None*	Not recorded			Rt. paratracheal	Lymphadenitis; lymphoma; cystic teratoma
No. 4 (H. S. W.)	23	M	W	Pain and burning over lower precordium	Neg	+++	Neg	Rt. posterior mediastinum adjacent to esophagus	Esophageal tumor
No. 5 (W. A. S.)	24	M	W	None	+	++	Neg	Rt. paratracheal	Cystic teratoma
No. 6 (G. N. H.)	22	M	N	None	Neg	Neg	Neg	Rt. paratracheal	Lymphoma; bronchogenic cyst
No. 7 (F. M. H.)	22	M	N	Hemoptysis	Neg	++++	Neg	Rt. paratracheal	Lymphoma
No. 8 (M. C. F.)	26	F	W	None	Neg	Neg	Neg	Left midmediastinum between aorta and pulmonary artery	Lymphoma
No. 9 (Z. D. L.)	31	M	W	*None*	+	Neg	Neg	Right midmediastinum extending around hilus	Lymphoma
No. 10 (A. C. T.)	23	M	W	None	Neg	++++	Neg	Rt. paratracheal	Mediastinal cyst
No. 11 (A. M. C.)	19	F	W	None	Neg	Not recorded		Rt. posterior mediastinum centered at level of carina	Teratoma
No. 12 (A. M. P.)	21	M	W	None	Neg	++++	Neg	Left midmediastinum level of carina	Lymphoma

abundance while in the other 3 cases the visualization of diagnostic forms required persistent microscopic search. Despite careful study of the other 8 mediastinal granulomas no fungi could be found. Particular attention was devoted to the "tuberculoma" previously purported to contain acid-fast bacilli, for the tuberculin skin test had been negative in that case and we, in our reinvestigations, had been unable to corroborate the presence of acid-fast organisms. Therefore, as a final maneuver, tissue sections from this lesion were also restained by the methenamine-silver nitrate technique of Gomori.[18] A few scattered Histoplasma were then discernible. In addition, a small granulomatous focus excised from the lung immediately adjacent to this particular lesion was found to contain many Histoplasma (Fig. 3), thus indicating a primary complex type of histoplasmosis having both a parenchymal and a mediastinal component. Such an experience suggests that, whenever a similar mediastinal tumor is removed, any associated parenchymal focus should also be excised as a possible aid in diagnosis.

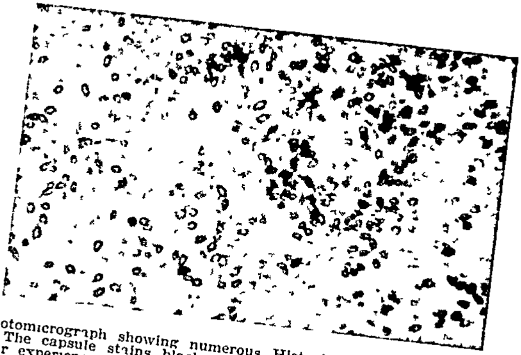


Fig 3.—Photomicrograph showing numerous Histoplasma (Gomori's methenamine-silver nitrate stain). The capsule stains black with the chromatin mass often remaining inconspicuous. In our experience this has been the most satisfactory method for demonstrating organisms, being more sensitive, less capricious, and affording better contrast than the PAS and Gridley techniques. Another advantage is that the methenamine-silver nitrate method includes a decalcifying process, so that calcium granules which in other stains present confusing artifacts, resembling yeast forms of *H. capsulatum*, stain only a light brown or not at all.

Thus, of twelve mediastinal granulomas, five were due to histoplasmosis (Figs 4-8). Two other cases were associated with positive skin tests to histoplasmin (Cases 4 and 10) but were histologically negative for fungi. Perhaps significant is the fact that in neither of these two specimens were there any zones of caseous necrosis, which in one instance (Case 10, Fig. 9) was due to evacuation of the tumor's necrotic contents in the process of excision and fixation of the specimen, while in the other (Case 4, Fig. 10) the pathologic process appeared "burnt out" and consisted mainly of fibrosis. There is the possibility, then, that these latter granulomas also represented residual histoplasmic foci, although in the final analysis we have grouped them among the nonspecific granulomas. Among the other granulomas, one (Case 6) presented a distinctive strongly positive histoplasmin reactors and, finally, (5) routine failure to establish a specific cause other than in that one case in which acid-fast bacilli were supposed to have been demonstrated Prompted by previous experience in demonstrating fungi in various culturally-negative granulomatous foci,[16, 17] the tissue blocks from each of these cases were restudied in order to see what part histoplasmosis might play in their etiology.

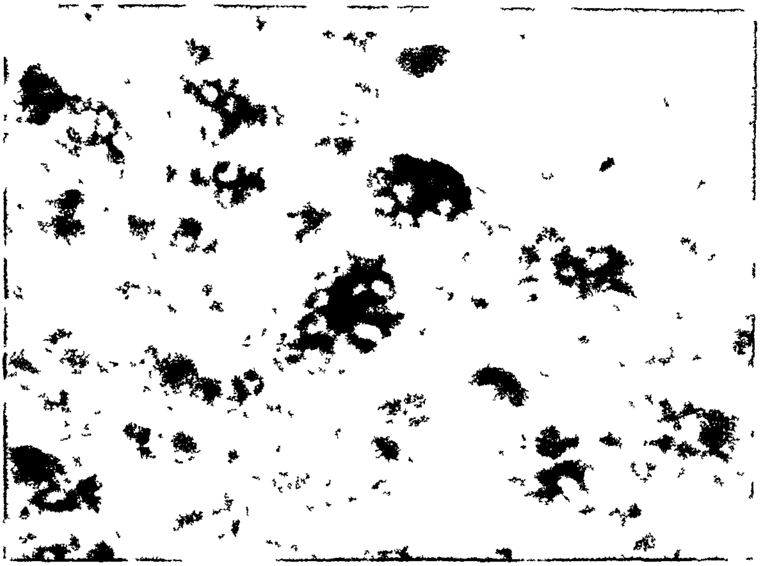


Fig 1 —High-power photomicrograph of the necrotic center of the mediastinal granuloma resected in Case 4 (periodic acid-Schiff [PAS] stain) Innumerable typical Histoplasma are seen, some filling the cytoplasm of macrophages, others lying free in the surrounding debris These organisms, none of which were identifiable in hematoxylin and eosin stained tissue sections, measure 3 to 5 microns in diameter and present a thin outer capsule Many contain a small crescent-shaped mass of chromatin material usually situated eccentrically and staining intensely red by the PAS method

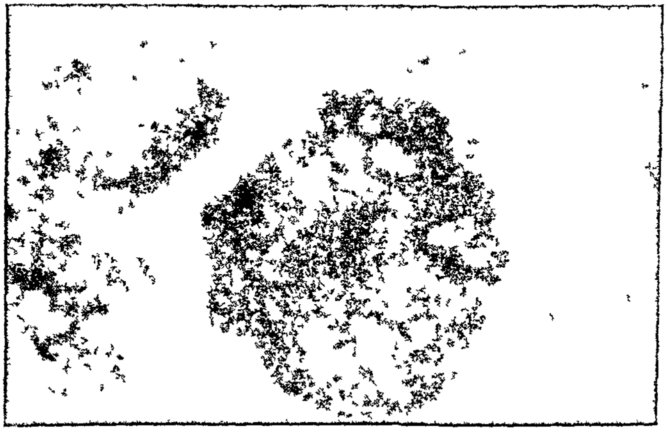


Fig 2 —Photomicrograph showing a small cluster of Histoplasma lying within a degenerated macrophage. The faint outline of additional, less distinct, yeast forms can be seen outside the cell In some instances a prolonged search may be required before finding diagnostic forms

Utilizing the periodic acid-Schiff (PAS) stain it was possible to demonstrate Histoplasma in 4 cases. Organisms when seen were invariably found in areas of caseous necrosis (Figs. 1 and 2). In one granuloma they were present in

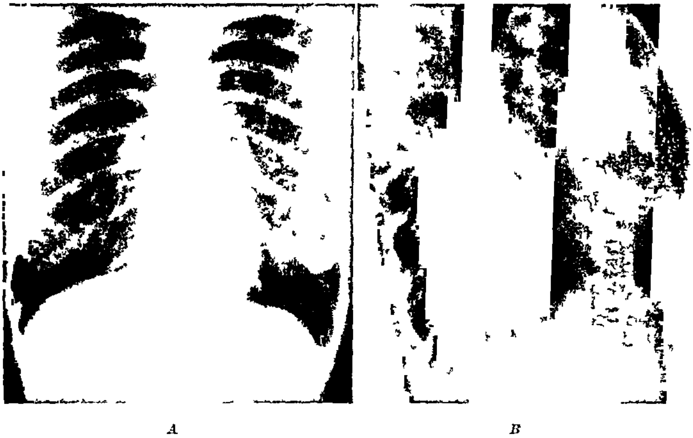


Fig 7—Case 3 A 27 year old white woman without any symptoms whatever Past history and physical examination were negative At thoracotomy a cystic tumor filled with thick white *material* was dissected *off* the trachea and *superior* vena cava This was misdiagnosed as a bronchogenic cyst Histoplasma were demonstrated later Note *the small* calcific parenchymal lesion seen in the lateral view just behind the sternum

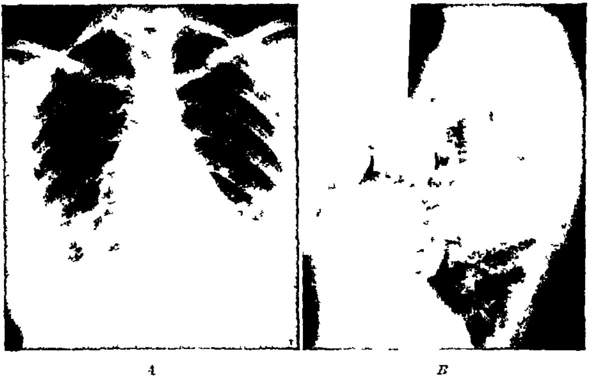


Fig 8—Case 11 A 19 year old asymptomatic white girl Routine posteroanterior chest roentgenogram (*A*) showed a large round tumor mass projecting from the right hilus Lateral x ray (*B*) localized it to the mid mediastinum beneath the carina The tuberculin skin test was negative The *resected cyst contained thick creamy material* and a few acid fast bacilli were reported to have been found in its caseous contents *An adjacent subpleural granuloma* was also excised from the right lower lobe Meticulous restudy failed to reveal any Histoplasma by the PAS method but neither could the presence of acid fast bacilli be corroborated by Ziehl Neelsen technique Utilizing the methenamine silver nitrate stain a few forms diagnostic of *Histoplasma* were *found in the mediastinal* granuloma while myriads of similar organisms were demonstrated in the associated pulmonary lesion (see Fig 3)

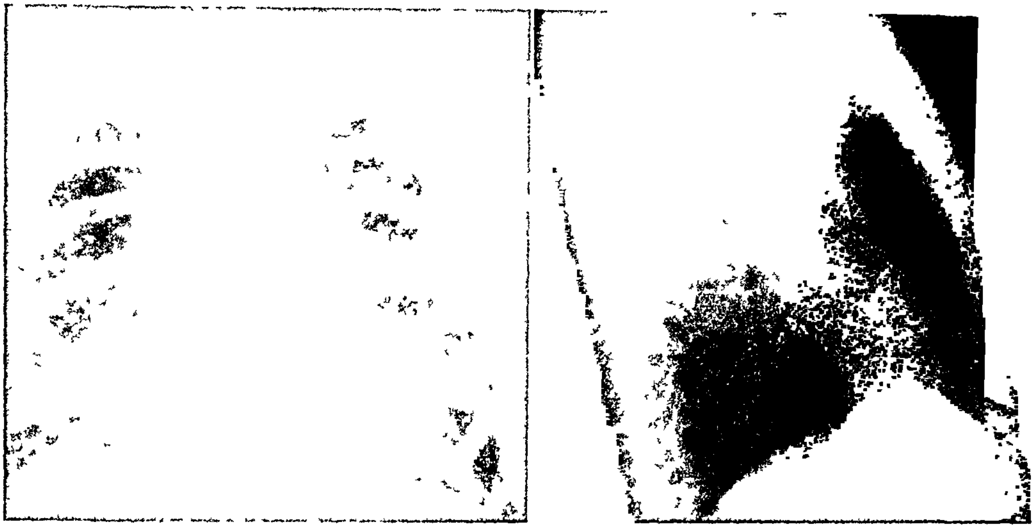


Fig 4.—Case 5 A 21-year-old asymptomatic white man whose right paratracheal lesion was discovered in a routine chest x-ray. Both tuberculin and histoplasmin skin tests were slightly positive. A cystic granuloma was resected, the original pathologic diagnosis being "tuberculoma," the final one being histoplasmic granuloma

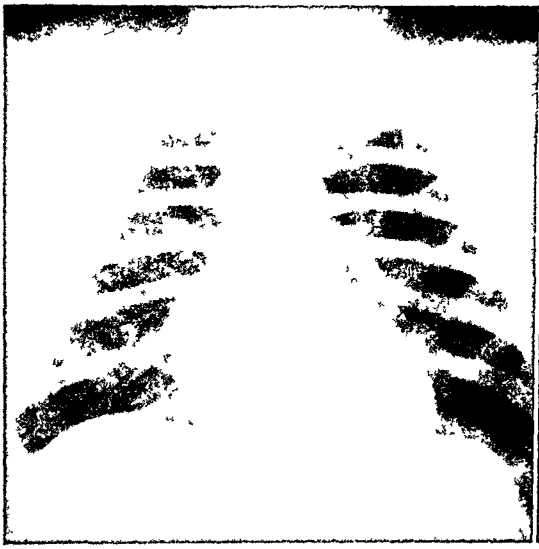


Fig 5.

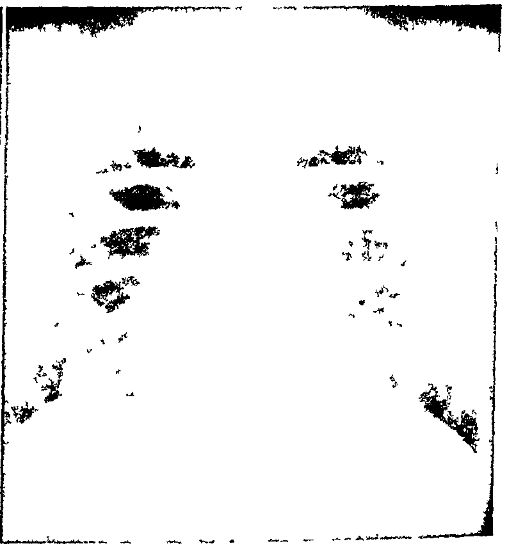
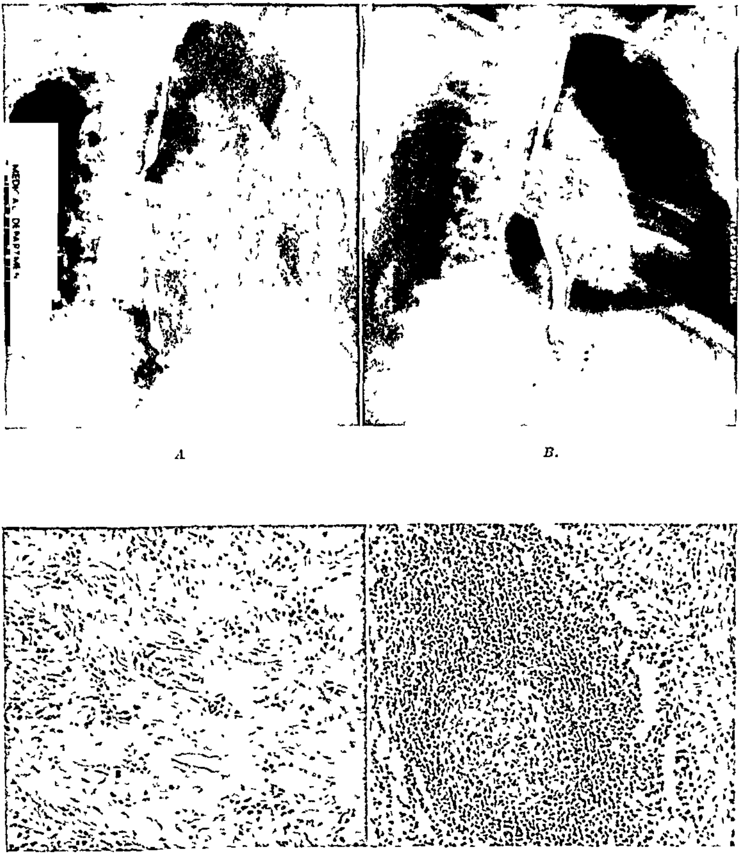


Fig 6.

Fig. 5.—Case 7 A 22-year-old Negro admitted to the hospital because of recent hemoptysis Chest roentgenogram disclosed a right paratracheal mass All preliminary studies were negative except for a strongly positive histoplasmin skin test. At operation a granuloma was encountered, initially diagnosed as a "tuberculoma" but later found to contain Histoplasma

Fig 6.—Case 12 A 21-year-old asymptomatic white man who in a routine x-ray was found to have a left hilar mass. With the exception of a strongly positive histoplasmin skin test all other investigations were negative. This lesion was also considered a "tuberculoma" until subsequent study by special staining methods revealed typical Histoplasma.



A

B.

C

D

Fig 10.—*Case 4* A 23-year-old white man whose only complaint was a burning sensation over the lower precordium. Examination was negative. Tuberculin skin test was negative; histoplasmin was strongly positive. Barium swallow demonstrated a smooth compression of the mid-esophagus thought to be consistent with a benign intramural esophageal tumor (*A* and *B*) At operation a firm mass of fibrous tissue was encountered. Microscopic examination revealed a large amount of dense collagenous connective tissue (*C*). Scattered throughout were small focal collections of chronic inflammatory cells and carbon particles. (*D*) Lymphoid follicles are seen presenting a prominent germinal center surrounded by a cuff of mature lymphocytes, the follicles being surrounded by a mass of connective tissue. This picture is characteristic of sclerosing mediastinitis (R Shuman, M.D., Armed Forces Institute of Pathology). No fungi were seen with [illegible]

type of reactive lymphoid hyperplasia (Fig. 11) which may cause the lesion to be mistaken for a thymoma, a danger emphasized by Castleman[19] and Iverson.[20] Another contained noncaseating granulomas typical of sarcoidosis (Fig. 12), while in another (Fig. 13) the polarizing microscope revealed a large number of doubly refractile particles, 1 to 3 micron in length, typical of silica crystals.

It seems quite logical to assume, therefore, that a certain percentage of mediastinal granulomas are the result of healed or healing mycotic infections. If the two can be considered comparable, one might infer that the mediastinal "tuberculoma" like the pulmonary "tuberculoma," is more commonly due to histoplasmosis and coccidioidomycosis than to tuberculosis. Thus, among 98 pulmonary granulomas analyzed by one of us,[21] Histoplasma were found in 53 (54 per cent), Coccidioides in 5 (5 per cent), and tubercle bacilli in 15 (15 per cent). The fact that cultures were consistently negative for fungi is probably a reflection of the "burnt out" character of these lesions. This is supported by the failure of the organisms to stain by routine methods, which in itself is a sign of nonviability. On the other hand, it may be necessary to use special cultural techniques similar to those used by Emmons and his co-workers,[22, 23] who obtained cultural proof that in at least one endemic zone 50 per cent of apparently normal dogs show histoplasmic involvement of mediastinal lymph nodes. Obviously, it is a common infection. The question now arises as to how frequently may such fungal involvement resolve so completely as to leave no radiographic and minimal anatomic evidence of previous histoplasmic lymphadenitis.

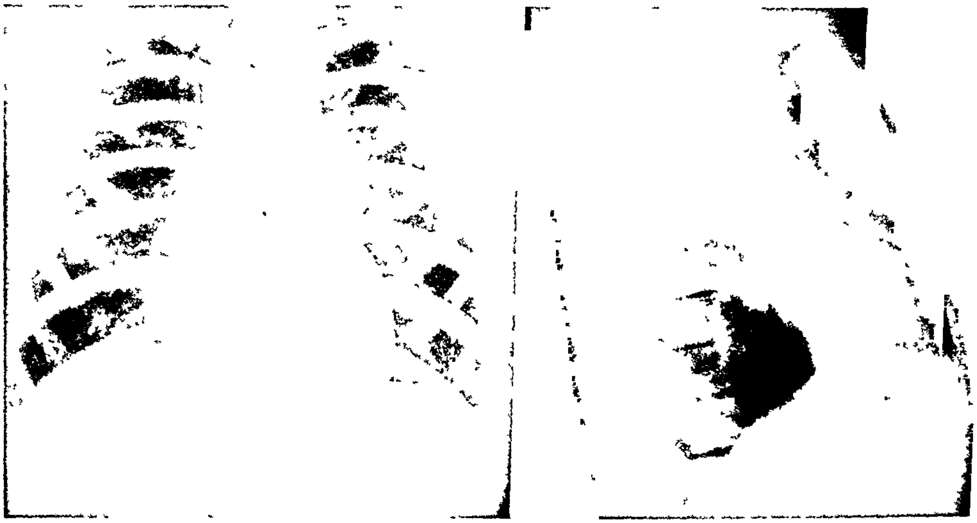


Fig. 9.—Case 10. A 23-year-old white man who had no symptoms. Histoplasmin skin test was strongly positive The resected granuloma occupied a characteristic location alongside the trachea and superior vena cava to which it was adherent. Re-examination of the specimen showed that it was not a dermoid cyst as had been originally reported but a granuloma instead. Unfortunately its pasty contents had been evacuated, thus obviating a specific diagnosis.

SUMMARY

Of 37 primary mediastinal tumors in patients operated upon at the U. S. Naval Hospital, Bethesda, Md., 12 were granulomas, a relative incidence far higher than that generally ascribed to this lesion. Two of these had been previously misdiagnosed, one having been called a cystic teratoma and the other

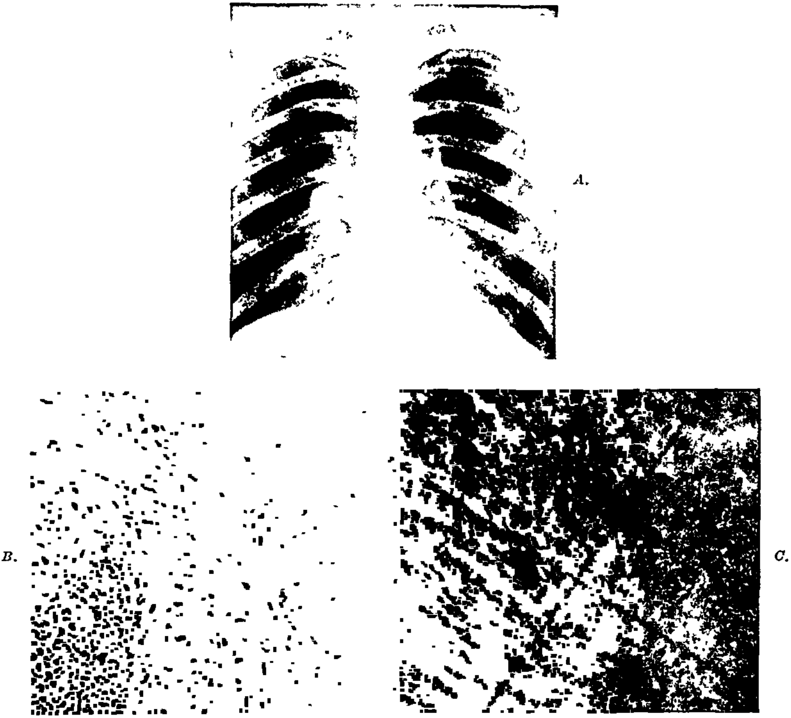


Fig. 13.—Case 9 A 31-year-old white man who presented with an asymptomatic mass in the right hilus (*A*) A large mass of matted lymph nodes were encountered at thoracotomy. Microscopically, there were prominent groups of lymphocytes surrounded by epithelioid cells. These are separated by a layer of fibroblasts from areas of fibrillary eosinophilic material *in which there are a few* blood vessels, *macrophages, and a large* amount of carbon *pigment* (*B*) With polarized light the central pigmented areas are found to contain many small (1 to 3 microns) doubly refractile particles resembling silica (*C*).

Fig 11—Case 6 A 22-year-old Negro without any symptoms related to right paratracheal mass (*A*). The resected specimen presented many lymphoid follicles with germinal centers containing large acidophilic masses as well as the usual large reticulum cells (*B*). With differential staining, these masses are identified as collagen Cuffs of small mature lymphocytes surround the germinal centers. The endothelial lining cells of the sinusoids are swollen, giving the appearance of small groups of epithelial cells with a lymphoid stroma *Superficially this lesion has some resemblance to a thymoma.*

Fig 12—Case 8 A 26-year-od asymptomatic white woman whose chest x-ray showed a left hilar mass (*A*). Microscopic examination of the resected specimen disclosed many granulomas varying in size and presenting a thin laminated layer of fibroblasts at the periphery surrounded by chronic inflammatory cells (*B*). Epithelioid cells and giant cells *are noted,* but *no areas of caseous necrosis are seen. Since no organisms could be* **demonstrated**, this lesion falls into that group of granulomatous reactions called Boeck's

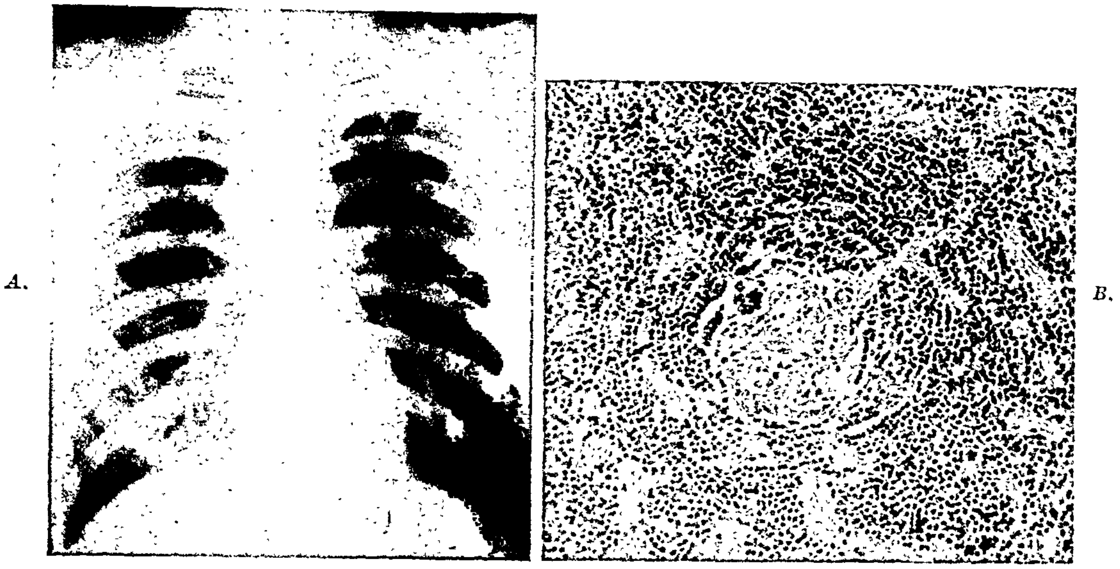


Fig. 11, *A* and *B*. *(For legend see opposite page.)*

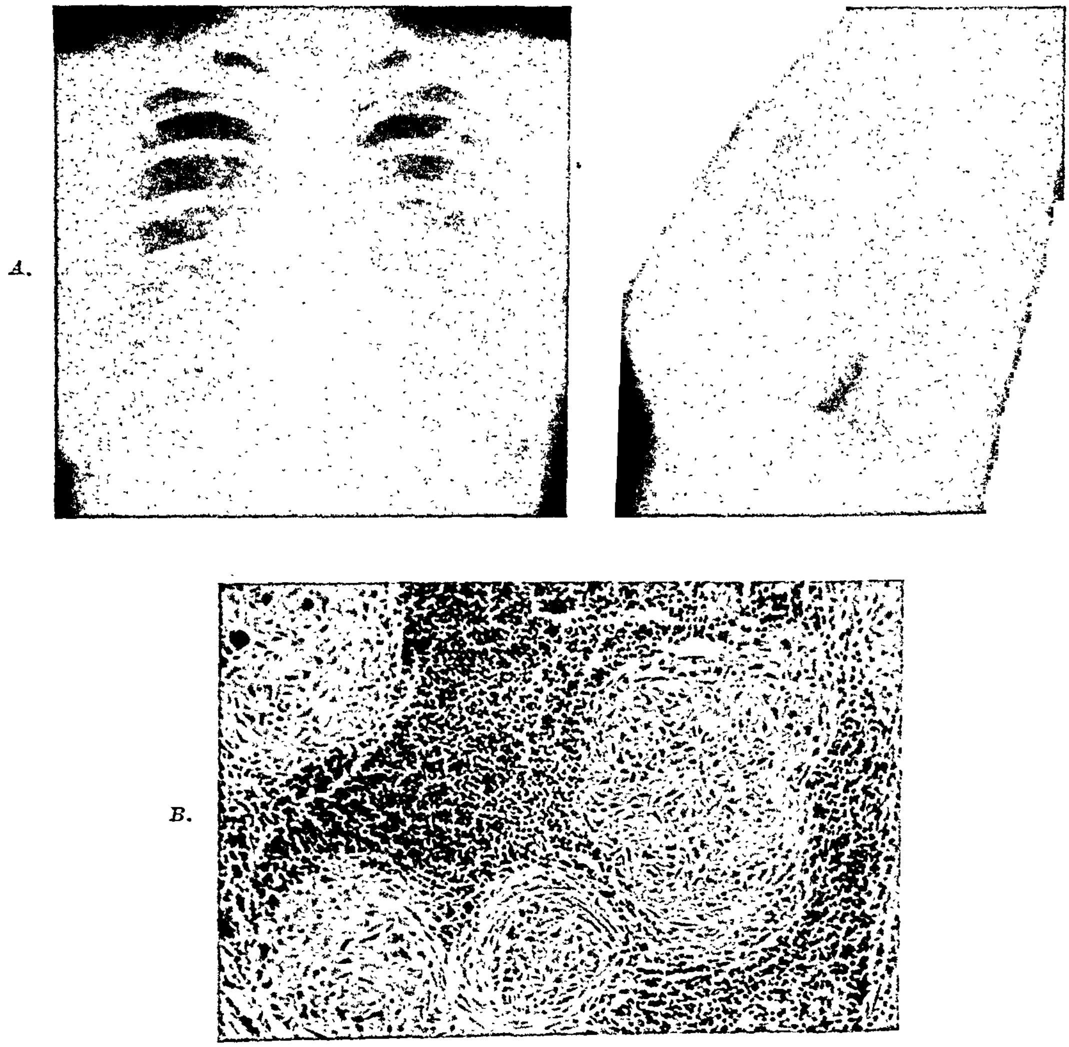


Fig. 12, *A* and *B*. *(For legend see opposite page.)*

TUBERCULOMA OF THE MEDIASTINUM

Report of the First Culturally Proved Case

J Winthrop Peabody, Jr, M D, Washington, D C, Harry E Walkup, M D, Oteen, N C, and James D Murphy, M D, Baltimore, Md

INCREDIBLE though it seems, a review of the literature on resected mediastinal tuberculomas fails to reveal a single instance in which tubercle bacilli could be cultured from the specimen.[1] Furthermore, in only one case were acid fast bacilli demonstrated histologically.[2] For this reason Samson and his co workers[3] and later Kunkel, Clagett, and McDonald[4] expressed doubt that these lesions were necessarily due to tuberculosis and preferred to call them granulomas in deference to their unknown etiology. Recent studies show that histoplasmosis accounts for a certain number of mediastinal granulomas and it has been suggested that the mediastinal granuloma like the pulmonary granuloma is much more commonly due to fungi than to *M tuberculosis*.[1] In the following case which appears to be unique, the resected specimen was teeming with tubercle bacilli and cultures were readily positive for *M tuberculosis*.

CASE REPORT

R B C, a 53 year old white veteran, was admitted to the Veterans Administration Hospital, Oteen, N C, for excision of a mediastinal tumor discovered during his recent hospitalization at VA Hospital, Mountain Home, Tenn, where he had been treated for a peptic ulcer.

The patient denied any respiratory symptoms other than a slight unproductive cough of approximately 5 years' duration. Past history and system review were not significant. Physical examination was not remarkable. Tuberculin (PPD No 1) and histoplasmin skin tests were both positive.

Multiple roentgenograms of the chest revealed a large round partly calcified mass in the mid mediastinum beneath the carina. The density measured about 8 cm in diameter and although obscured in the posteroanterior projection by the cardiac silhouette, it was readily visualized in the lateral and oblique views as well as in tomograms (Fig 1). There was widening of the carina with pressure on the right stem bronchus. A small calcific density was noted in the anterior basal segment of the right lower lobe. The roentgenologist's impression was a teratoid tumor or a mass of calcified tuberculous lymph nodes.

Operation.—With the patient in the left lateral decubitus position under endotracheal cyclopropane oxygen anesthesia, the right pleural space was entered through the bed of the subperiosteally excised sixth rib. After dividing the inferior pulmonary ligament and retracting the lung upward, a firm tumor could be palpated beneath the carina anterior to the esophagus. The right vagus nerve was adherent to the mass and was divided. By a process of sharp and blunt dissection the tumor was freed from the right inferior pulmonary vein, right stem bronchus, and then from the carina and left stem bronchus, to all of which structures it was adherent. The entire tumor was removed, and the chest closed. Postoperative course was uneventful and the patient remains well after a course of antituberculous chemotherapy.

From the Surgical Service Veterans Administration Hospital Oteen N C

Received for publication May 21 1957

a bronchogenic cyst. Most of the others were presumed to be tuberculomas, although cultures had been uniformly negative and acid-fast bacilli were demonstrated in only one.

Re-examination of these granulomas by the periodic acid-Schiff stain has demonstrated Histoplasma in 4 cases. In still another case, Histoplasma were found only after resorting to Gomori's methenamine-silver nitrate stain. Two other lesions associated with strongly positive histoplasmin and negative tuberculin skin tests were negative for fungi as were the other 5 mediastinal granulomas.

It is suggested that histoplasmosis is more commonly the cause of mediastinal granulomas than has previously been thought.

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Pathology—Grossly, the tumor was a smooth, well encapsulated mass measuring 6 cm in diameter and weighing 190 Gm It felt somewhat mushy except for one surface which presented a firm calcified exterior On cut section the tumor was found to be filled with white caseous material resembling tooth paste (Fig 2) The wall of the cyst was quite thin measuring 1 mm in thickness

Microscopically, the capsule of the cystic tumor was comprised of hyalinized fibrous tissue containing many islands of calcification some of which actually represented bone and contained marrow spaces Scattered throughout the wall were small collections of lymphocytes In one area there was distinct lymphoid structure containing anthracotic pigment There was no epithelial lining

Direct smears of the caseous contents reveal a great number of acid fast bacilli Cultures were positive for *M tuberculosis*

SUMMARY

A mediastinal tuberculoma is reported in which there was ample histologic and bacteriologic proof of its tuberculous origin As far as the authors can determine this is the first such case to be reported

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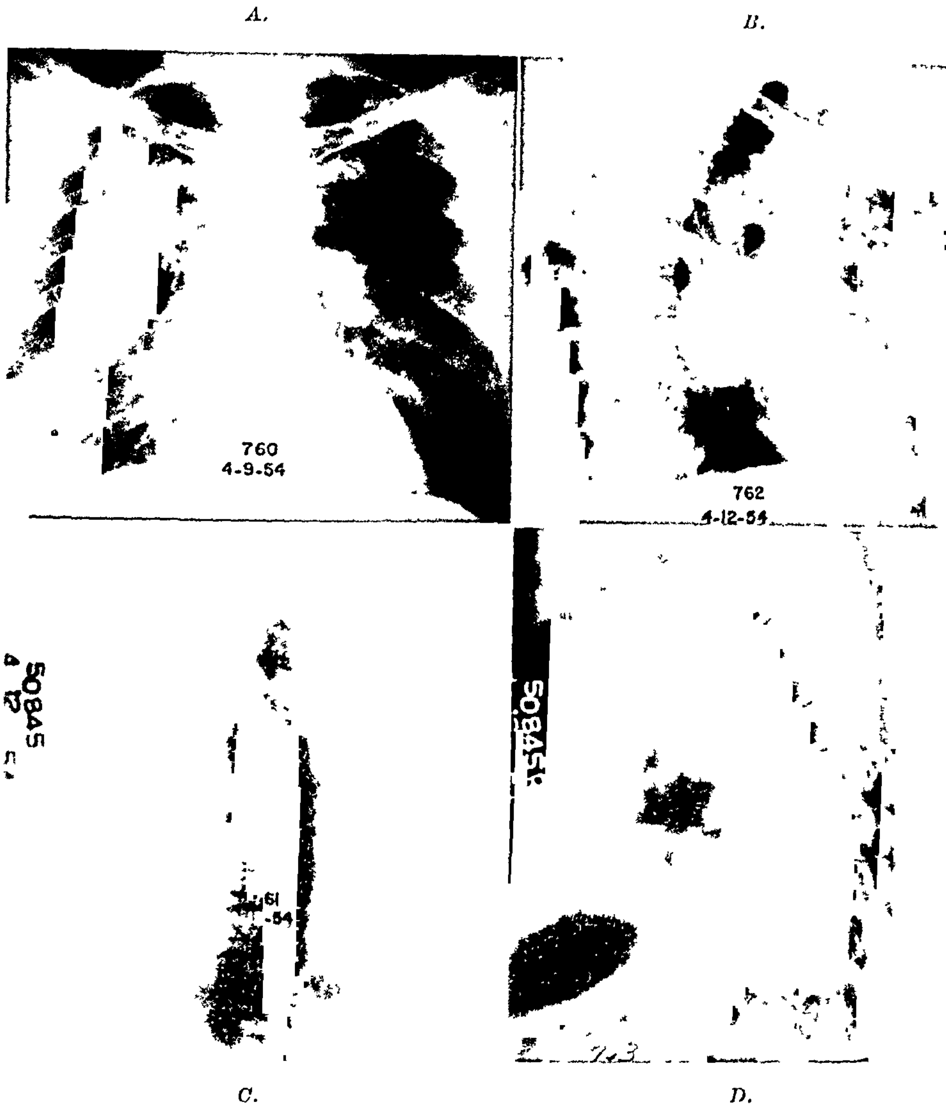

Fig. 1.—Chest roentgenograms showing the round partly calcified mid-mediastinal mass. Only the right margin of the tumor is seen in the posteroanterior projection (*A*) but oblique (*B*) as well as overpenetrated posteroanterior (*C*) and lateral (*D*) views show it quite clearly.

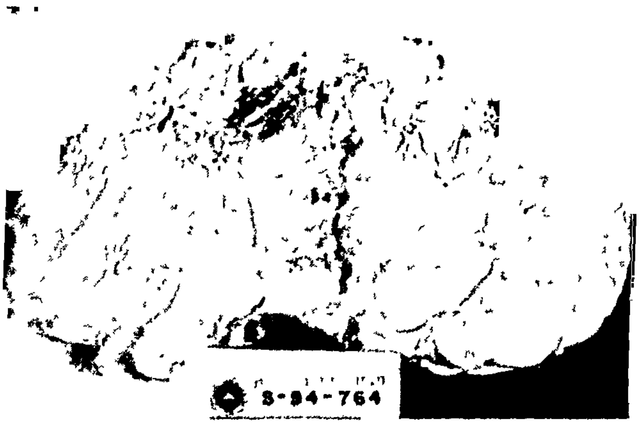

Fig. 2.—Photograph of the resected specimen. Cut surface reveals the "toothpasty" contents which were teeming with acid-fast bacilli on smear and from which *Mycobacterium tuberculosis* was readily recovered by culture.

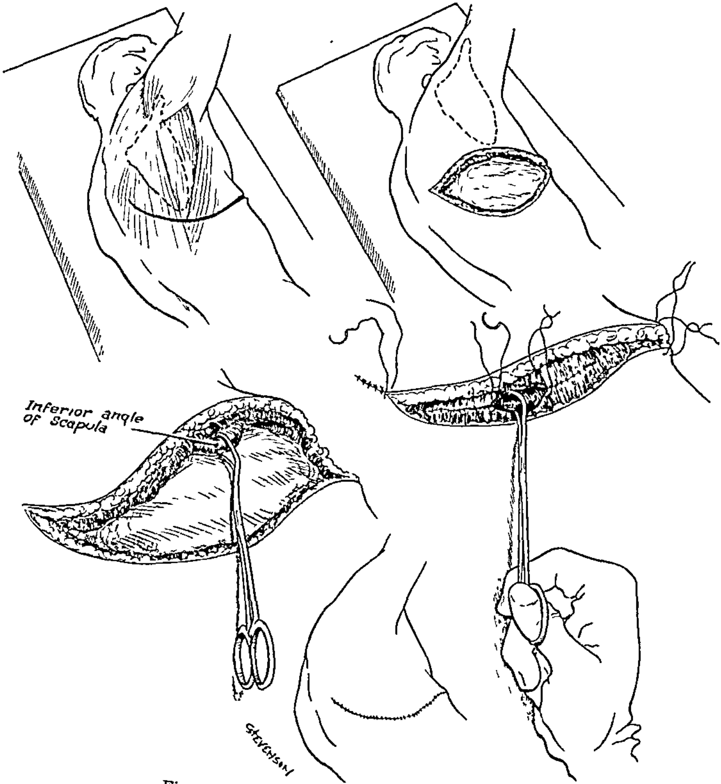

Fig. 1.—See text for explanation.

A SIMPLE METHOD OF SCAPULAR TRACTION FOR CLOSURE OF LATERAL OR POSTEROLATERAL CHEST INCISIONS

O. C. Brantigan, M.D., and P. S. de Borja, M.D.
Baltimore, Md.

OCCASIONALLY there is developed a simple, easy, but different method of doing some common, everyday process or task, which is worthy of reporting. In closing a lateral or posterolateral thoracic wound, the task of holding the shoulder in place is a difficult one for the assistant or for the anesthetist. If the shoulder is not held in proper position the muscles and skin do not lie properly for accurate reapproximation. It has been found by repeated practice that once the ribs have been approximated the inferior angle of the scapula, covered by its normal muscles, can be brought easily into view. An ordinary towel clip, clamped through the muscles into the bone of the inferior angle of the scapula, provides secure control of the shoulder (Fig. 1). With one hand it is easy for the assistant to keep the shoulder in proper position by traction on the towel clamp, leaving the other hand free for other tasks. The incision is unobscured, making approximation by suture simple and easy. Removal of the towel clamp is at times slightly awkward and therefore the muscles must not be sutured too closely to the towel clamp before it is removed.

SUMMARY

In the closure of a lateral or posterolateral thoracic incision a towel clamp applied to the inferior angle of the scapula easily provides the necessary traction for holding the shoulder in the proper position while the tissues of the wound are being accurately reapproximated by suture.

From the Department of Surgery, Church Home and Hospital, Baltimore, Md.
Received for publication May 10, 1957.

Clinically, pulmonary cryptococcosis most often simulates a low-grade subacute or chronic pneumonitis, tuberculosis, neoplasm, or lung abscess.[1, 5, 8] Chest roentgen findings usually fall under one of the following categories as described by Bonmati and his co-workers.[9]

1. Large, solitary rounded mass or several nodular lesions which may excavate. This includes "coin lesions," "toruloma," or pseudo-tumors.
2. Disseminated nodular lesions of miliary or slightly larger size, frequently with linear infiltrates.
3. Single or multiple infiltrates which may be either patchy or linear in character.

There is no dependable skin test. Diagnosis may be proved by identifying the organisms in the sputum, pleural fluid, or bronchial aspirations, by their isolation in cultures grown in Sabouraud's medium at 37° C., and establishing their pathogenicity by inoculation into mice or guinea pigs.[1, 12] The use of special tissue staining techniques, such as the periodic acid-Schiff, Gomori methenamine-silver preparation, and the mucicarmine stain, is essential.[11, 16, 31] Ordinary hematoxylin and eosin sections may show the organisms as poorly defined, yeastlike structures with occasional budding, located mostly intracellularly in macrophages or giant cells, or extracellularly in necrotic centers of the granulomas. The special stains mentioned give sharper definition so that the organisms are more readily identified. In the experience of the Armed Forces Institute of Pathology, with the exception of occasional Blastomyces, the cryptococcus is the only one of the more commonly encountered fungi that takes the mucicarmine stain.[16, 31]

INDICATIONS

In the absence of central nervous system involvement, we believe that all patients with localized pulmonary cryptococcosis are definite candidates for excision of their pulmonary lesions, unless they are suffering from other hopeless systemic disease, or are not acceptable surgical risks. Localized pulmonary disease is interpreted to mean that the lesion or lesions do not involve more than one lobe of either lung, or not more than one lung, if the lesion is multilobar. The more localized the lesion in the lung, the greater the indications for excision. Although less ideal, the indications may be extended to include exceptional cases with very well-localized bilateral nodular or "coin" lesions which do not involve more than one lobe of both lungs, so that bilateral pulmonary resection is still possible. Upon fulfillment of the above criteria, pulmonary resection should not be delayed. Lobectomy is the procedure of choice if the lesion is unilobar. Wedge or segmental resection is less desirable because of the greater likelihood of incomplete removal of a lesion caused by organisms which have a great tendency to invade the central nervous system. Pneumonectomy is indicated in the presence of multilobar unilateral disease in which all lobes of one lung are involved.

Should adequate preliminary diagnostic studies fail to reveal the nature of a localized pulmonary lesion which may be of cryptococcal [illegible] in, the [illegible] n

RESECTION IN PULMONARY CRYPTOCOCCOSIS (TORULOSIS)

Manuel White, M.D., and L. S. Arany, M.D.
Walla Walla, Wash.

CRYPTOCOCCOSIS (torulosis, European blastomycosis, Busse-Buschke's disease, 1894) is a subacute or chronic, very often fatal, infection caused by *Cryptococcus neoformans,* a neurotrophic, yeastlike, nonsporulating, nonmycelial, budding fungus, 5 to 15 micra in diameter, characterized by a wide gelatinous capsule in India ink stain.[1-6, 31] The organism is of world-wide distribution, affecting both sexes and all ages.[43, 46] It should be differentiated from *Blastomyces dermatitides,* the cause of Gilchrist's disease, isolated in 1898,[1, 6, 7, 39, 40] and *Histoplasma capsulatum,* originally described by Darling in 1906.[49, 50] The infection is either localized or systemic. The organisms show a great predilection for the central nervous system, with manifestations of a subacute or chronic meningitis which is still almost invariably fatal.[1, 7, 8] The disease is not transmitted from man to man, from animal to animal, or from animal to man. The infection is believed to be air-borne from a reservoir in the soil.[31] The most common portals of entry appear to be the respiratory tract and the skin. Dissemination from a localized focus in the lung, skin, or other organs takes place through the blood stream and lymphatics. Small lesions in the lungs may heal by fibrosis and eventual calcification.[1, 5, 9, 32]

Primary cryptococcosis is not too uncommon, with more than 310 cases now on record in world's medical literature, and nearly 38 fatal cases a year recorded by the National Office of Vital Statistics during recent years.[8, 9, 25, 26, 28-31, 38] At least 100 reported cases had clinical manifestations of a pulmonary disease sometime during their courses, and approximately one third of these showed localization to one lobe or lung.[8, 9, 27] Since Dormer and his colleagues,[10, 48, 51] in 1945, first reported a case of localized pulmonary cryptococcosis treated by resection, there have been at least 25 additional cases, including our own, bringing the total to 26 cases reported, treated in this manner.[9, 11-15, 23, 24, 33, 35, 37, 41, 42, 48]

DIAGNOSIS

The diagnosis of pulmonary cryptococcosis of localized form seldom has been made before operation. There is reason to believe that the diagnosis has been missed not infrequently after operation and even after autopsy. A more routine use of special histologic staining techniques in every case of obscure pulmonary disease, particularly "coin lesion," "chronic granuloma," and those considered to be "resolving pneumonia" or "non-specific pneumonitis," will most likely result in the diagnosis of more cases.[11-13, 31]

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closely approximated the lesion originally seen. On Dec. 22, 1954, there was further enlargement and apparently complete coalescence of these two lesions, thereby forming a large area of uniform haziness at the mesial aspect of the right mid-lung field. On lateral planigrams the lesion appeared to be wedge-shaped, mainly involving the superior segment of the right lower lobe; however, the posterior and lateral basal segments appeared to be involved also. By Jan. 3, 1955 (Fig. 1), there was a definite decrease in the size of the area of uniform density, especially at its parahilar aspect.

Bronchoscopy, on Dec. 20, 1954, was negative except for a moderate amount of white sputum from the right lower lobe. A right anterior scalene lymph node biopsy was negative. It was felt that bronchogenic carcinoma could not be ruled out.

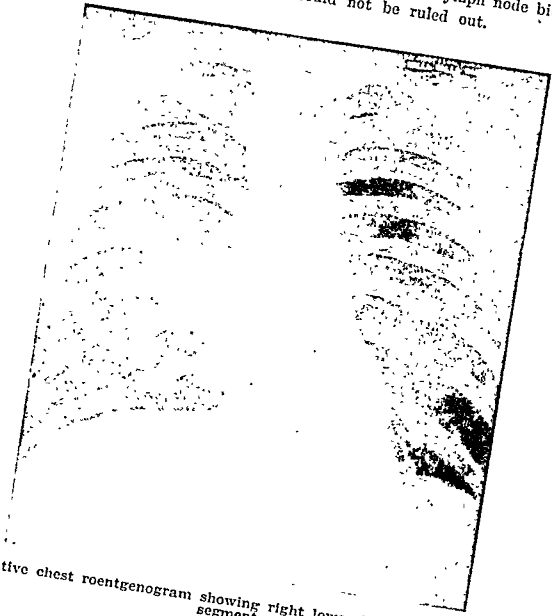

Fig. 1.—Preoperative chest roentgenogram showing right lower lobe lesion, mostly in superior segment.

On Jan. 14, 1955, a right exploratory thoracotomy was performed, combined with biopsy and frozen section studies from two nodular lesions within the right lower lobe, followed by a right lower lobectomy. The report on frozen sections was that of a granulomatous or inflammatory lesion.

Gross examination of the excised right lower lobe revealed a firm, atelectatic superior segment which contained multiple abscesses filled with a thick yellow liquid. There were other patchy areas of consolidation within the medial and posterior basal segments, and at least two other small, hard, calcified nodules in the remainder of the basal segments. Microscopic report by the Armed Forces Institute of Pathology, Washington, D. C., indicated a granulomatous lesion (Figs. 2 and 3) containing many yeastlike organisms with occasional budding, mostly within giant cells and macrophages. The organisms were well defined by the Gomori methenamine-silver preparation. Mucicarmine stain demonstrated the organisms to be carminophilic. The diagnosis was pulmonary cryptococcosis.

should then be considered an indeterminate one. Unless there are overwhelming contraindications such as those already alluded to, the presence of an indeterminate pulmonary lesion is an indication for an exploratory thoracotomy and excision.

Since at present there is no entirely satisfactory medical treatment for the almost invariably lethal cryptococcal meningitis, in spite of iodides, actidione, stilbamidine, modern chemotherapeutic agents and antibiotics.[5, 7, 15, 17-20, 31] excision of a localized pulmonary lesion must be done early, before the development of meningitis or central nervous system disease. Approximately 75 per cent of the patients in 100 reported cases of pulmonary cryptococcosis eventually died with central nervous system involvement.[9, 10, 28] Resection for localized pulmonary cryptococcosis should be considered in the same sense as resection for bronchogenic carcinoma, as proposed by Taber,[21] Froio and Bailey,[14] Berk and Gerstle,[13] and Poppe.[33, 47] In Australia, where cryptococcosis is better recognized, pulmonary lesions are resected, not only as a prophylaxis against nervous system involvement, but also in the hope that resection will affect the course of the disease more favorably even when meningitis is already present. Clinical improvement and apparent recovery have been reported following resection in the presence of cryptococcal meningitis in at least 2 cases by McConchie[37] and Susman.[36, 52] The follow-up periods have been 6 months and about 5 years, respectively. Susman's patient received postoperative roentgen therapy to the skull and entire spine, with a total skin dose of 6,900 r, given expectantly.[52, 53] Cryptococcal meningitis or meningoencephalitis is known for its chronicity and long periods of remission.[44, 45]

CASE REPORT

A 37-year-old white male Army veteran was admitted to the Veterans Administration Hospital, Walla Walla, Wash., on Dec. 16, 1954, with complaint of right chest pain for the previous 3 months. A survey roentgenogram of the chest with a portable unit had revealed the presence of a lesion in the right lung approximately 3 months before the patient came to the hospital. The patient had received a course of antibiotics just prior to admission, prescribed by a local physician for treatment of what was believed to be a right pneumonia. Low-grade fever had been present intermittently since the onset of the right chest pain. The patient had smoked one and a half packages of cigarettes daily for many years. The only positive physical findings on admission consisted of some restriction of inspiratory expansion of the right side of the chest and the presence of harsh breath sounds over the right upper half of the chest. Râles were absent.

Laboratory data on entry showed a hemoglobin of 13.2 Gm., white blood cell count of 13,800, with 72 per cent neutrophils. Blood sedimentation rate was 21 mm. The intradermal tuberculin PPD first and intermediate strengths, coccidioidin, and histoplasmin tests were negative. PPD second strength was weakly positive. Repeated sputum examinations for acid-fast organisms on smear and culture were negative.

A miniature survey film of Sept. 24, 1954, revealed a small area of confluent infiltration at the third anterior interspace on the right, and a subsequent film of Oct. 19, 1954, showed no essential change. A film of Dec. 6, 1954, disclosed a small central highlight within the area of infiltration and, at the same time, marked enlargement of the right hilus. On Dec. 13, 1954, there was further increase in the right parahilar density, which now

The postoperative course was entirely uneventful The patient was sent home on the twelfth postoperative day He soon returned to work and has remained entirely well Follow up films of the chest in the second postoperative week and 17 months after lobectomy (Fig 4) revealed no recurrence The patient remains entirely well 28 months after the operation, and is now working in Anchorage, Alaska

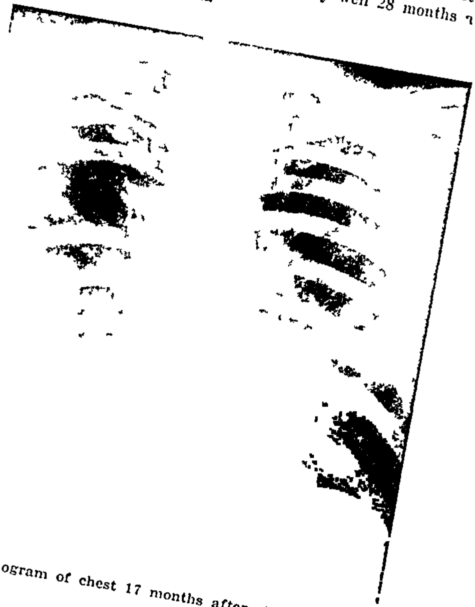

Fig 4 —Roentgenogram of chest 17 months after right lower lobectomy

RESULTS AND DISCUSSION

Table I shows the reported cases of resection for localized pulmonary tococcosis in available literature

Significant is the fact that 19 of the 21 reported survivors or app cures had no central nervous system manifestations prior to surgical exc of localized pulmonary lesions[9,14,33,35,37,41,4] The importance of long follow up of resected cases before cures can be claimed was well illustrated the case reported by Palmrose and Losli[23] Their patient developed cryptoco meningitis more than 3 years after enucleation of a right lower lobe lesion a died during the following year One can only conjecture whether or not t course of the disease would have been altered more favorably if a lobectom instead of a simple enucleation, had been performed

SUMMARY AND CONCLUSION

1 Of the 26 reported cases of sufficiently localized pulmonary cryptococcosis treated by resection, 21 (81 per cent) survived free of symptoms an

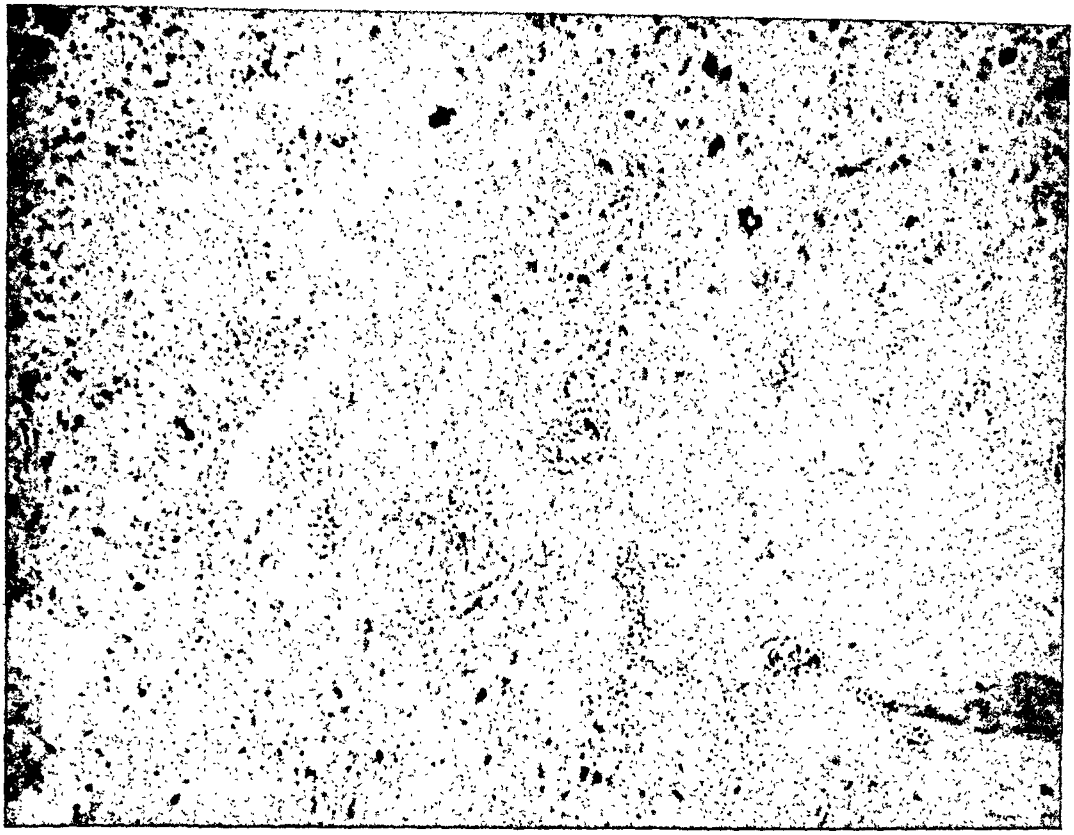

Fig. 2.—Low-power microscopic view of granulomatous appearance of lesion with giant cells. (Hematoxylin and eosin stain ×100; reduced ⅙.)

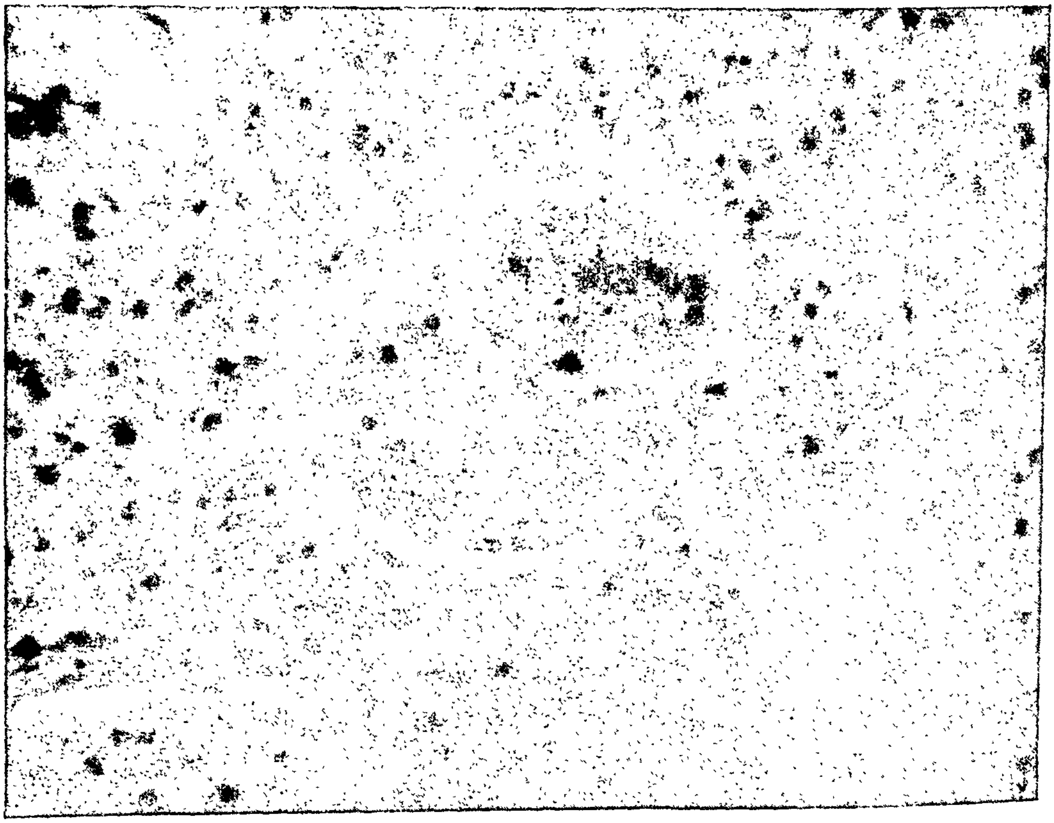

Fig. 3.—High-power microscopic view of giant cell laden with white, yeastlike *Cryptococcus neoformans.* (Hematoxylin and eosin stain ×430; reduced ⅙.)

4 A cure cannot be claimed justifiably unless the patient has remained well and without roentgenographic evidence of recurrence for at least 4 years following resection

5 The early surgical excision of a localized pulmonary lesion before the onset of central nervous system dissemination can be regarded not only as a preventive, but also as a curative procedure

6 The early adequate surgical excision of localized pulmonary cryptococcosis without central nervous system involvement appears to be the only effective treatment now known for this disease

ADDENDUM

More recent communication reveals that all three resected cases reported by Ian McConchie of Australia, in 1951, remain alive and well Among these, the man who had a left lower lobectomy, in 1940, represents the longest reported known survivor or cure, 17 years The woman who was treated by excision of the pulmonary lesion and the adjacent chest wall in the presence of cryptococcal meningitis recovered and remains well 6½ years later The third patient is alive and well 8 years after left lower lobectomy The number of patients who have already remained well 4 years or more following pulmonary resection among the 21 reported survivors is, therefore, raised to at least 9

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Table I

AUTHOR AND YEAR REPORTED	CASES	APPARENTLY CURED SURVIVORS	RESULTS
Dormer et al., 1945 and 1947	2	1	One alive and apparently well 3 months later, in spite of postoperative c.n.s. disease which responded to large doses of iodides; other died with c.n.s. disease 31 days later, in spite of iodides
Starr and Geddes, 1949	1	1	Alive and well 7 years later
Froio and Bailey, 1949	1	1	Alive and well more than 13 years later
McConchie, 1951	3	3	One asymptomatic for 10 years with undiagnosed small circumscribed lesion in contralateral lung; other with c.n.s. disease before resection improved and apparently well 6 months later; another alive and well 2 years later
Palmrose and Losli, 1952	1	0	Died with c.n.s. disease 4 years later, with onset of symptoms more than 3 years after enucleation of lesion
Cruickshank and Harrison, 1952	1	0	Died with pulmonary embolism and c.n.s. disease 6 days later
Berk and Gerstle, 1952	1	1	Alive and well more than 8 years later
Baker, 1952	3	3	Alive and well 9 months to 4 years later
Schepel and Carsjens, 1953	1	1	Alive and well 2 months later
Susman, 1954	2	2	One alive and apparently well almost 5 years later, in spite of c.n.s. disease before resection; other alive and well 5 years later.
Poppe, 1954	2	2	Both alive and well 18 months later
Nichols and Martin, 1955	1	1	Alive and well 18 months later
Bonmati et al., 1956	4	3	Three alive and well 4 months to 2 years and 8 months later; one died of pneumonia and c.n.s. disease within a year later
Wettingfield et al., 1956	1	0	Died with small bowel obstruction, carcinoma of testis with metastasis, and pulmonary tuberculosis 5 weeks later
Pinney, 1956	1	1	Alive and well 1 month later
White and Arany, 1957	1	1	Alive and well 30 months later
Totals	26	21	

are apparently cured. The preceding table of results presents a range of apparent cures for periods of 1 month to more than 13 years.

2. Since 17 per cent of the reported resected cases of patients without prior central nervous system involvement subsequently developed it, the prognosis remains uncertain.

3. The most favorable prognosis is limited to cases in which there is no evidence of central nervous system involvement before excision of the localized pulmonary lesion; 90 per cent of the apparently cured survivors fall under this category.

A STAINLESS STEEL BUBBLE OXYGENATOR

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THERE are three main types of extracorporeal blood oxygenators: membrane,[2, 5] film,[1, 3, 6, 8, 9] and bubble.[4, 7, 10] We wish to record a bubble type oxygenator which is made entirely of stainless steel. The advantages are in refinements. It can be entirely assembled before autoclaving, it is indestructible, and it does away with the debubbling heating helix. It can efficiently oxygenate volumes of blood from 100 c.c. to 4,000 c.c. per minute while adequately removing carbon dioxide to maintain the pH in a safe range.

DESCRIPTION

The oxygenator is constructed from two stainless steel tubes joined a common debubbling chamber (Fig. 1). The length of these tubes is 1 cm and the oxygenator tube is 1⅜ inches in inside diameter. The blood is loaded through a side vent, which is ½ inch in inside diameter, for gravity feed or it can be made ¼ inch for pressure feed. The oxygenator manifold is a stainless steel disc containing 250 holes. These openings are .0024 inch in diameter. The filter manifold is a funnel-shaped disc of stainless steel which will act as a seat for the stainless wire mesh blood filter of the Baxter blood-giving sets.

The debubbling chamber is a 4,000 c.c. stock gauze container (Fig 2). A fitted wire basket holding stainless steel sponges is inserted. No debubbling substances are necessary to make the removal of carbon dioxide adequate.

The second vertical tube is 1 inch in inside diameter and, functionally, goes only to the bottom of the debubbling reservoir, at which point four openings have been placed. This tube is a blood reservoir in which the blood is heated by a flood lamp to maintain a normal temperature. The blood hold-up in this tube is 500 c.c. Blood is pumped from the bottom of the manifold chamber after passing through the filter to return to the patient. These two tubes, although having different inside diameters, are interchangeable in their seats in the debubbling chamber.

The oxygen manifold chamber and filter manifold chamber are interchangeable (Fig. 3). Thus, blood can be oxygenated in the short tube with small inside diameter or in the larger, longer tube in large volumes.

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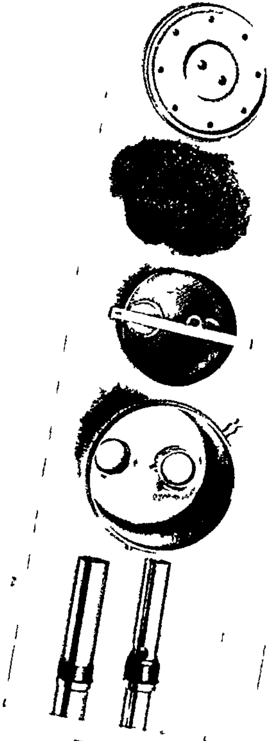


Fig 2

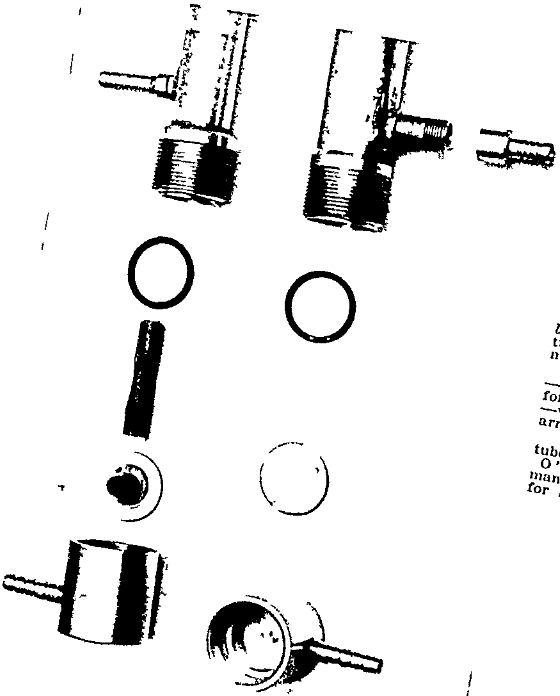
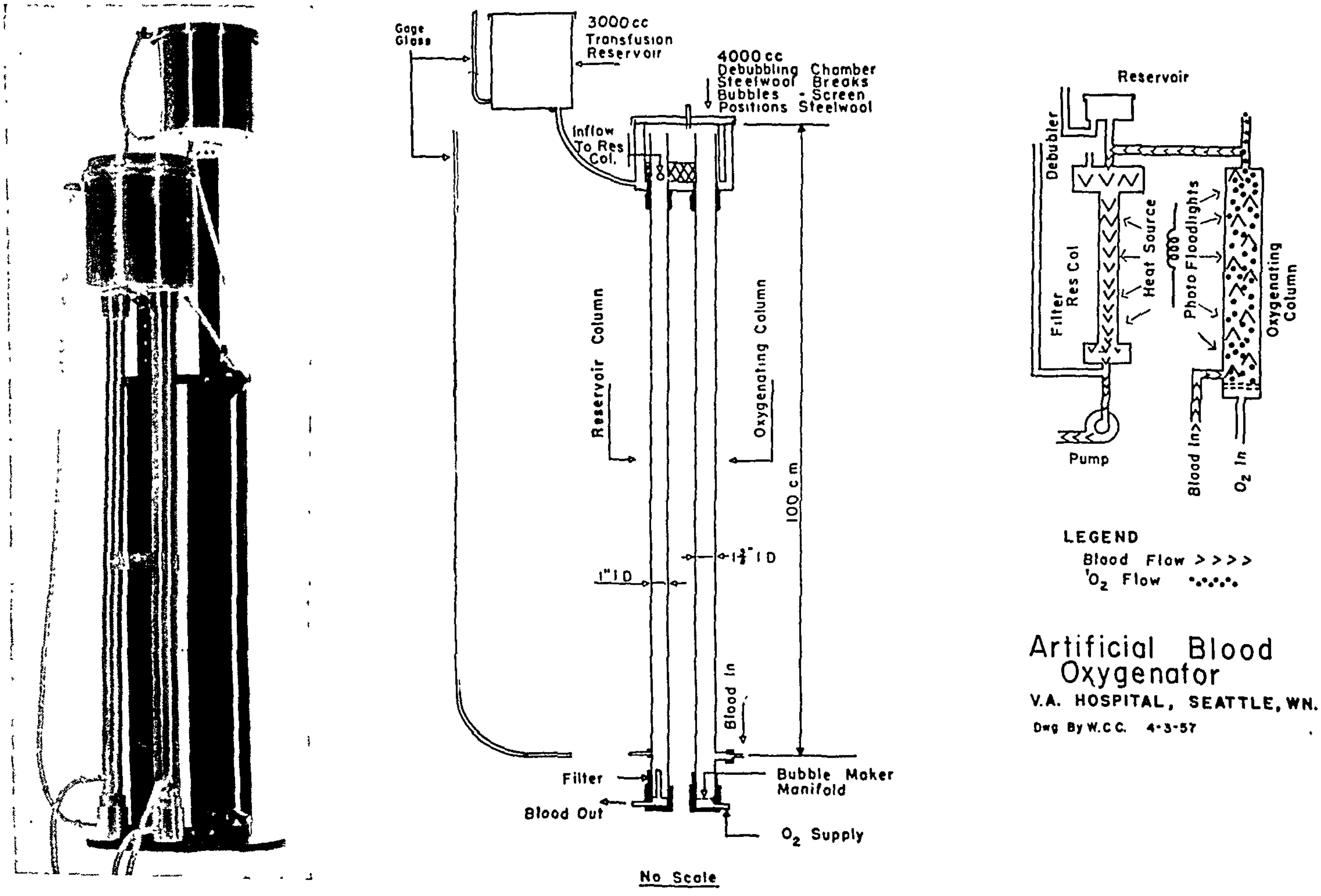


Fig 3

Fig 2—Debubbling chamber *From bottom to top*—Oxygenating and reservoir tubes, debubbling chamber, stainless steel mesh holder stainless steel mesh and cover

Fig 3—*Right side, from bottom to top*—Oxygen manifold chamber with side arm for oxygen inlet oxygen manifold O ring—washer and oxygenating column and side arm with reduction nipple

Left side, from top to bottom—Reservoir tube, with side tube for sight tube attachment, O' ring— washer stainless steel filter, filter manifold and filter chamber with side arm for blood withdrawal



A.

B.

Fig. 1.—*A*, Oxygenator. *B*, Diagram of oxygenator.

TABLE III

20 30 MINUTE BY PASS

	EXP NO	DOG, BEFORE BY PASS	OXYGENATOR, BEFORE BY PASS	DOG, END OF RUN	OXYGENATOR, END OF RUN
Platelets	1	337 000	50 000	150 000	150 000
	2	300 000	60,000	150 000	
	3	290,000	70 000	150 000	160 000
	4	184 000	94 000	88 000	88,000
Free hemoglobin mg /100 c c	1	26	65	56	46
	2	80	112	32	32
	3	4	4	170	170
Hematocrit	1	48	44	46	
	2	44		52	48
	3	44	41	52	48
	4	42	45	44	43
	5	41	45	47	45
White blood count	1	9 700	8 300	9 000	11 500
	2	8,200	8 600	9 000	9,500
Fibrinogen mg /100 c c	1	280	260	216	210
	2	340	290	230	330
	3	260	240	160	190
	4	250	217	240	240

CONCLUSION

A simple two vertical tubed oxygenator constructed completely of stainless steel is reported It meets the requirements of simplicity with adequate performance

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The level of blood in the blood reservoir tube can be seen in the sight glass (Fig. 1).

A stainless steel blood reservoir of 3,000 c.c. is connected to the bottom of the debubbling chamber by plastic tubing for rapid transfusion. This reservoir can be raised or lowered to either transfuse or bleed the system.

A suction tube nipple is provided in the top of the debubbling chamber. Thus, blood of the suction flow is not lost but retransfused into the oxygenator.

PERFORMANCE

This oxygenator oxygenates at different minute outputs adequately (Table I).

TABLE I

EXP. NO.	RATE OF FLOW /MIN. C.C	HEMOGLOBIN		OXYGEN SATURATION IN VOLUME PER CENT		OXYGEN SATURATION PER CENT	
		VENOUS	ARTERIAL	VENOUS	ARTERIAL	VENOUS	ARTERIAL
1	500	17.30	17.10	7.58	22.34	32.21	94.61
2	1,000	17.60	17.25	13.84	21.92	57.84	92.69
3	1,500	17.61	17.35	12.42	23.51	52.67	99.18
4	2,000	17	16.75	18.13	22.79	78.71	99.24
5	3,000	16.45	16.5	17.98	21.63	80.67	95.57

The carbon dioxide loss as reflexed in the pH during perfusion can be shown to be adequate (Table II).

TABLE II

DOG NO.	TIME IN MINUTES OF PERFUSION	DOG BEFORE RUN		OXYGENATOR BEFORE RUN	OXYGENATOR	
		VENOUS	ARTERIAL		VENOUS	ARTERIAL
59	0	7.44	7.53	7.40		
	10				7.44	7.50
	25				7.45	7.54
	35				7.46	7.52
	45				7.44	7.52
	60				7.42	7.49
	73				7.45	7.52
58	0	7.46	7.56			
	5				7.45	7.55
	15				7.46	7.55
	19				7.44	7.54
57	0	7.60	7.71	7.39		
	5				7.43	7.64
	10				7.53	7.61
	15				7.52	7.61
	20				7.51	7.61
53	0	7.45	7.49			
	6				7.44	7.49
	14				7.40	7.49
	25				7.39	7.49

All values represent pH.

The effects on the formed and unformed elements of the blood are detailed in Table III.

The top edge is held flush with the lower edge of the inverted "V" in the bottom of the bars by a 5 mm. stainless steel rod welded into the base of the "V," the edge of the screen being bent to fit between it and the bar. In assembling the unit, stainless steel shims measuring 0.15 mm. (0.006 inch) in thickness are placed at each end of bars, providing a 0.15 mm. by 30.5 cm. slit for blood to be admitted from the top chamber to the screens. The shims and bars are bolted together by 2 rods at each end. The use of shims between the bars

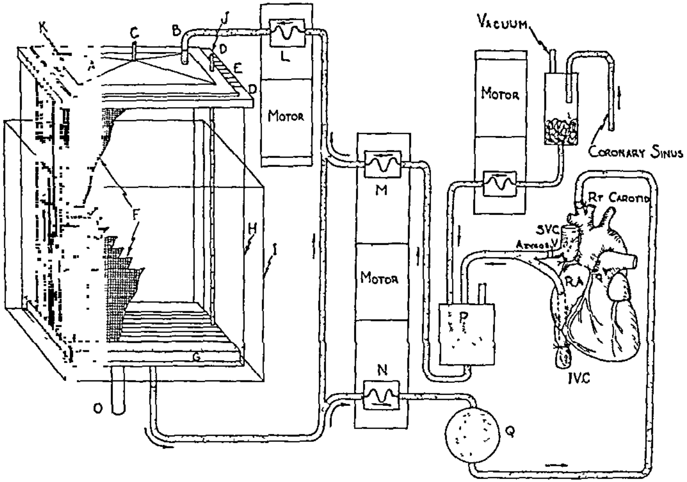

Fig. 1.—Schematic diagram of the pump-oxygenator. See text for description.

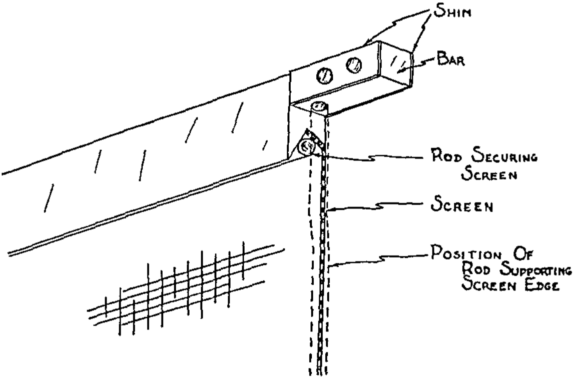

Fig. 2.—Diagram of bar and method utilized to attach screens.

THE USE OF A MODIFIED GIBBON-TYPE PUMP-OXYGENATOR

I. Description of Apparatus

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THE successful employment of a screen-type oxygenator for use in an extracorporeal circulation has been demonstrated by Gibbon[1] and Kirklin.[2, 3] However, the high cost and engineering problems related to the construction of the model they employ have been a deterrent to its general use. Because of these factors, a modified version of the "Gibbon" stationary battery-type screen oxygenator has been constructed in such a manner as to eliminate costly and difficult fabrication methods without significantly altering its oxygenating function.

DESCRIPTION OF APPARATUS

Basically, the unit resembles that described by Gibbon[1] (Fig. 1). The top chamber (*A*) is constructed of Plexiglas in a pyramidal shape, its inside dimensions measuring 7.4 by 30.4 cm. with a height of 1.25 cm. The blood enters this chamber through inlet *B*. Tube *C* allows air to be removed from the chamber during filling, evacuation of any air which may enter the system during perfusion, and the addition to the system of any drugs desired. Soft rubber gaskets are placed on both sides of the plastic top chamber and the whole unit bolted through a rectangular steel cover, allowing securing of the chamber to the floor and tightening of the floor to the outside case.

The floor of the chamber is formed by 9 stainless steel bars which extend beyond the plastic top chamber, the 2 outer bars, *D*, measuring 2.5 by 37.5 cm., the 7 inner bars, *E*, 0.9 by 37.5 cm. The top and sides of these bars are machined flat, the bottom surface is shaped to form an inverted "V" along their length. The screens are attached along the length of the bar to one side of this "V" (Fig. 2).

The 8 stainless steel screens, *F*, measuring 30.5 by 55 cm. are fabricated from Tyler No. 538 screen.† The greatest dimension of the rectangular mesh in the screens is oriented in the horizontal plane. The screens are supported on the sides and bottom by 5 mm. stainless steel rods welded with silver solder.

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†Tyler Wire Company Cleveland, Ohio.

emptied from the bottom outlet, the circulating pump being left on to continue the filming of saline on the screens until blood is added to the oxygenator.

Heparinized blood is added to the system through the venous reservoir, the remaining saline being emptied as the blood film forms on the screens and reaches the bottom of the oxygenator. The remainder of the system is then filled with blood, displacing the saline. Approximately 1,600 c.c. is required to prime the system, distributed as follows: 85 c.c. in the top chamber of the oxygenator, approximately 800 c.c. on the screens, 300 c.c. in the arterial reservoir in the bottom of the case, 300 c.c. in the venous reservoir, and 100 c.c. in the tubing and filter.

As soon as the blood is added and before placing the animal on the unit, a mixture of humidified 97 per cent oxygen and 3 per cent carbon dioxide is started at 10 L. per minute through the oxygen inlet tube.

METHOD OF PROCEDURE

Donor blood in sufficient quantity to prime the unit and replace expected blood loss is obtained for dog experiments by femoral cannulization after paralyzing the donor animal with 20 to 30 mg. of succinylcholine, and supporting respiration with intermittent endotracheal insufflation of room air. Heparin, 50 mg. per liter, aqueous penicillin, 1,000,000 U. per liter, and streptomycin 0.5 Gm. per liter are added at the time of bleeding.

Animals to be placed on extracorporeal circulation are prepared preoperatively by administration of morphine sulfate, 1.5 mg. per kilogram, atropine, 0.03 mg. per kilogram, and procaine penicillin, 600,000 U., given 45 minutes before the procedure is started. Pentothal sodium, 2.5 per cent, is administered intravenously for anesthesia and intermittent endotracheal insufflation of room air is utilized to maintain respiration throughout the procedure.

A routine right thoracotomy is performed, entering the pleural cavity in the fifth intercostal space. Meticulous care is taken to ligate all bleeding vessels. Tapes are placed about the superior and inferior venae cavae adjacent to their entry into the right atrium to be used for occlusion later. The right common carotid artery is isolated via an incision in the neck. A plastic catheter is passed into the carotid artery centrally. The superior and inferior venae cavae are also cannulated with plastic catheters inserted into the azygos vein and right auricle, respectively. Blood samples for pH, oxygen, and carbon dioxide contents are obtained at this time, at intervals during the procedure, and just after stopping the extracorporeal circuit.

The extracorporeal unit is then promptly connected to the animal, making certain no air bubbles are trapped in connecting the arterial cannula to the arterial outflow tube.

The arterial and venous pumps are now started, their flow rates previously set at 60 c.c. per kilogram per minute. The venous pump is speeded, maintaining the level in the venous reservoir until maximum venous return is obtained. Coincidentally the arterial pump is speeded, keeping the level of the permits the use of any number of screens desired, depending on the flow rate through the oxygenator. Those not in use are blocked by a single shim extending the length of the bar.

The base of the screens is placed in a Plexiglas block, *G*, the edges fitting into machined slots. The block is constructed so that it almost fills the internal dimensions of the base of the outside case, *H*, leaving approximately ⅛ *inch clearance on all sides so that minute changes in* blood volume in the reservoir produce significant changes in the height of the blood. One oxygen inlet tube, *J*, extends the length of the screens at one side of them and fits into the plastic block below while 3 oxygen outlet tubes, *K*, are placed at the opposite side; their length is such that oxygen flows out of the top, middle, and bottom of the unit. The tubes are press-fitted through the top bars.

The outside case, *H*, is constructed of ¼ inch Plexiglas, its dimensions such that it just allows insertion of the assembled screens, oxygen tubes, and base block, the bars resting on its top edge. A ¼ inch I.D. blood outlet tube and a ¾ inch O.D. saline outlet tube, *O*, are provided in the bottom of the case. A second case, *I*, is constructed outside case *H* to provide a thermostatically controlled water bath for constant control of the blood temperature and to prevent condensation of water vapor on the interior of the case.

Sigmamotor finger pumps* are utilized rather than the modified DeBakey pumps used in the originally described oxygenator. These pumps provide a pulsatile rather than a constant flow. Their speed is controlled manually with a vernier gear control.

These pumps are placed in relation to the oxygenator as shown in Fig. 1, pump *L* acting as a cycling pump for the oxygenator, and pumps *M* and *N* as venous uptake and arterial output pumps, respectively. A plastic venous reservoir (*P*) placed approximately 3 feet below the level of the right auricle receives blood by gravity flow from the venae cavae. Mayon plastic tubing† and stainless steel connectors are used throughout except for the short segments of soft latex tubing placed in the finger pumps.

The filter *Q* is constructed of two pieces of Plexiglas with fine mesh nylon cloth‡ held across the cavity between them. With the inflow placed at the top and the outflow at the bottom, the filter also acts as an air bubble trap.

This entire unit for extracorporeal circulation can be constructed and assembled for a cost of approximately $3,000.00, and eliminates many of the difficulties encountered in constructing the originally described models. Flow rates of 400 c.c. per minute for each screen can be obtained. This will give a total flow of 3.2 L. per minute if all 8 screens are used.

In operation, the entire unit is initially filled and washed with physiologic saline. The single Sigmamotor pump *L* which functions as the circulating pump for the oxygenator is used to fill the case completely with saline, its rate set to exceed the anticipated perfusion rate for the patient and to keep the top chamber filled, but not under pressure. The saline is then slowly

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Original Communications

CONDUCTION SYSTEM IN HUMAN HEARTS WITH INTERVENTRICULAR SEPTAL DEFECTS

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Improved techniques in cardiac catheterization and angiocardiography have resulted not only in early diagnosis of cardiac anomalies, but have provided accurate information as to the location, size, and number of developmental defects.[1, 2] The rapid advances in thoracic and open cardiac surgery have kept pace with the above improved diagnostic methods, so that attention has now been focused upon the need for more detailed knowledge of heart anatomy in certain cardiac anomalies.

Defects of the interventricular septum were of particular interest to the authors, for such defects are not an uncommon anomaly and have been closed surgically with success by several investigators.[3-6] The gross anatomy of such human septal defects was described by Lev[7] and classified clinically by Warden[8] and Becu[9] and their co-workers, yet little information was to be found on the intracardiac location of the specialized conducting tissue in hearts with such interventricular septal defects.[10] The need for such a microscopic study was further emphasized recently by Lillehei in the discussion of another paper,[6] when he stated, "the incidence of complete heart block has been about 12 per cent in the patients having ventricular septal defects repaired."

It should be pointed out that the development and microscopic structure of such specialized cardiac elements in the human heart have been studied.[11-14] The excellent histologic studies of Yater and co-workers[15, 16] were unique in that they studied in detail the conduction tissue of 9 human hearts to determine the pathogenesis of bundle branch block, but none of these hearts had septal defects.

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blood in the bottom of the oxygenator constant. This procedure is a relatively simple one since the large plastic block in the base of the case results in rather marked changes in the level with relatively small changes in volume. With proper adjustment of the pumps, the venae cavae are occluded by sliding rubber tubing tightly over the tapes and clamping. In most instances, endotracheal respiration is stopped during the perfusion. Blood may be returned to the venous reservoir and subsequently to the extracorporeal circuit by means of the coronary sinus system as illustrated in Fig. 1. Any blood loss is manifested by a fall in the level of blood in the venous reservoir.

The pH is followed throughout the perfusion and acidosis partially or completely corrected by the addition of 7.5 per cent sodium bicarbonate, usually in about 50 c.c. amounts. Protamine in amount equal to 1½ to 2 times the amount of heparin employed is then administered directly into the right auricle or very slowly intravenously during closure of the chest.

Both endotracheal and chest tubes are left in place until the animals show definite lightness of anesthesia, usually about one hour after the perfusion is terminated. In some of the animals with both weight loss and a fall in hematocrit after the procedure, citrated blood is given intravenously in adequate amounts.

During the initial runs of dogs on this pump oxygenator, during which time modifications were being made, the mortality was significant. With the final model as described above, 8 dogs have been operated upon. These animals had a right ventriculotomy and were maintained on the pump-oxygenator for 30 minutes. Of these dogs, 7 survived. The eighth dog was given an intravenous dose of protamine sulfate, penicillin, and streptomycin about one hour postoperatively. Shortly after this, it developed shock and died. A review of the physiologic changes in the experimental animals will be reported separately.

SUMMARY

A modified Gibbon-type pump-oxygenator has been described. This model functions well at high flows and can be constructed at a low production cost.

The authors wish to thank Mr. Severin Altpeter and Mr. Stanley Altpeter of the Puro Co., Inc., St. Louis, Mo., for the construction of the oxygenator described in this report.

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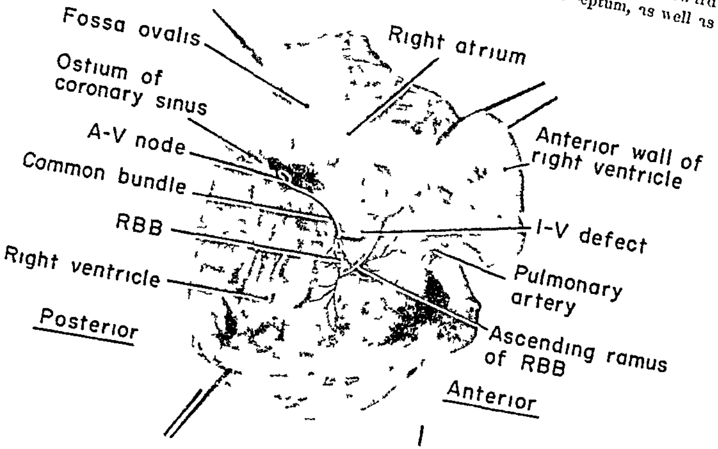
The large thin left bundle branch and its rami are illustrated in Fig 2 It should be noted that a small slender fascicle of the left bundle branch coursed under and then ascended anterior to the defect The main portion of the left branch continued toward the apex of the left ventricle and gave off numerous stout fascicles to the septum, as well as the anterior and posterior walls of the left ventricle



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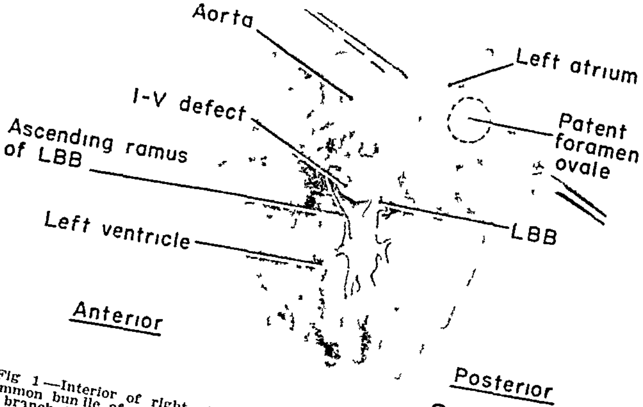

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Fig 1—Interior of right atrium and right ventricle of Specimen 1 Note relation of the common bundle of conduction tissue to defect within interventricular septum The right bundle branch (*RBB*) descended in the septal myocardium (dotted) and divided into ascending and descending rami as it reached the endocardium inferior to the defect The anterior and posterior surfaces of the heart are indicated in this and all of the following illustrations

Fig 2—Interior of left atrium and left ventricle of Specimen 1 The location of the thin broad left bundle branch (*LBB*) is shown as it descended in the posterior margin of the septal defect Note ascending ramus of left bundle branch

The present study was an attempt to provide more precise anatomic information on the location and course of the specialized conduction tissues of human hearts that had high interventricular septal defects.

MATERIALS AND METHODS

Fifteen human hearts with high interventricular defects were used in this study. All of the specimens had additional cardiac anomalies although no hearts in our series demonstrated any abnormal ante-mortem cardiac rhythm. There were 8 male and 7 female specimens; 14 were from patients 2 days to 3 years of age, while one was from a 27-year-old patient.

The gross specimens were photographed for future reference. The anterior wall of each was cut and marked with India ink for ease of orientation prior to tissue fixation and embedding. Nine were embedded in celloidin, one in paraffin, and 5 specimens were used for gross study under a dissecting microscope (see Figs. 15-18). The 10 specimens used for microscopic study were cut in serial section and stained individually by a Masson trichrome-light-green method. A wax model reconstruction was made of a 2-month-old specimen to demonstrate the position and relationships of the specialized conduction tissues to the septal defect (see Fig. 7).

RESULTS

The specialized tissues of the conduction system were easily identified in sections stained with the Masson technique by their pale stained appearance. All muscular elements were red in contrast to the light-green appearance of the connective tissue elements.

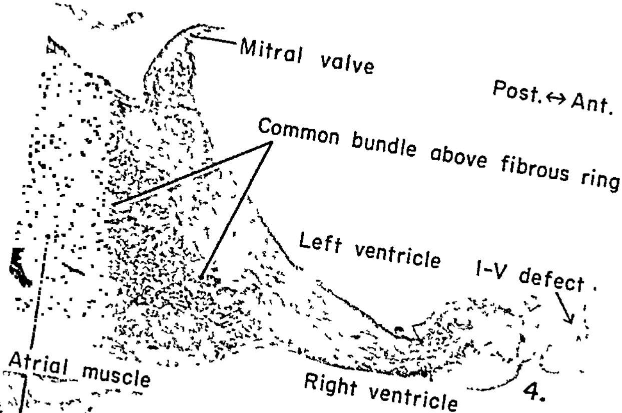
We were surprised to observe extensive hemorrhage in the specialized tissue of 6 of our specimens. Hemorrhage was noted within the A-V node and common bundle of 3 hearts, in only the common bundle of 2 specimens, and within the A-V node of another.

In all 10 specimens, we were able to trace the continuity of the specialized muscle fibers of the conduction system from the atrioventricular node to the common bundle with its left and right bundle branches. As the common bundle (of His, or crus) was followed distal to the fibrous ring, it was located in the posterior margin of the interventricular septal defect in all but 3 specimens. The location and mode of branching of the common bundle is best presented by a description of this structure in the individual specimens.

Specimen 1. (No. A 14-55, white female, aged 2 days.)

As shown in Figs. 1 and 2 this specimen had a thin translucent interatrial septum and patent foramen ovale, as well as a high interventricular septal defect. The location and course of the common bundle and its branches were inserted on the photographs following a study of the serial sections.

Note the position of the common bundle as it proceeded distally in the thin posterior margin of the defect to divide into left and right bundle branches at the top of the interventricular septum (i.e., at the postero-inferior margin of the defect). At the point of origin of the 2 bundle branches, many fascicles of pale stained fibers could be traced from the common bundle and its 2 branches into the more heavily stained muscle fibers of the interventricular septum (the paraspecific fibers of Mahaim[17]).



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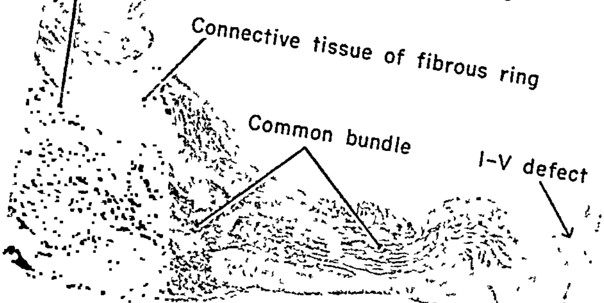

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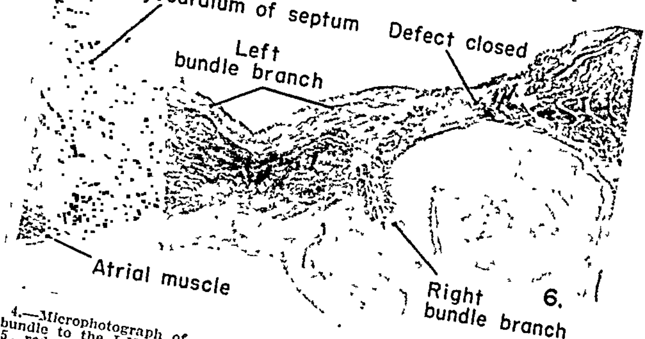

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Fig 4.—Microphotograph of a section of Specimen 2 Note the atrial position of the common bundle to the I-V defect before it passed through the fibrous ring. (Masson technique. ×25, reduced ⅔.)

Fig. 5—Microphotograph of Specimen 2 illustrating the relationships of the common bundle to the defect, as it crossed the fibrous ring (annulus fibrosus). (Masson technique. ×25; reduced ⅔.)

Fig 6—Microphotograph of Specimen 2 at the lower margin of the I-V defect as it closed The common bundle (Fig 5) has just divided into a thin left bundle branch and a more compact right bundle branch For orientation of Figs. 4, 5, and 6 see Figs. [illegible]d 7. (Masson technique; ×25; reduced ⅔.)

The slender right bundle branch descended within the interventricular septum to gain a subendocardial position part way down the right side of the septum as shown in Fig. 1. The right bundle branch also had a small but discrete fascicle of specialized fibers that ascended anterior to the septal defect (Fig. 1). Smaller twigs from the ascending ramus became lost in the myocardium of the septum anterior to the defect.

Specimen 2. (No. A 5774, white female, age 2 months.)

This specimen with a high septal defect (Fig. 3) was one of our most interesting cases, for the specialized elements of the conduction system were extremely well differentiated.

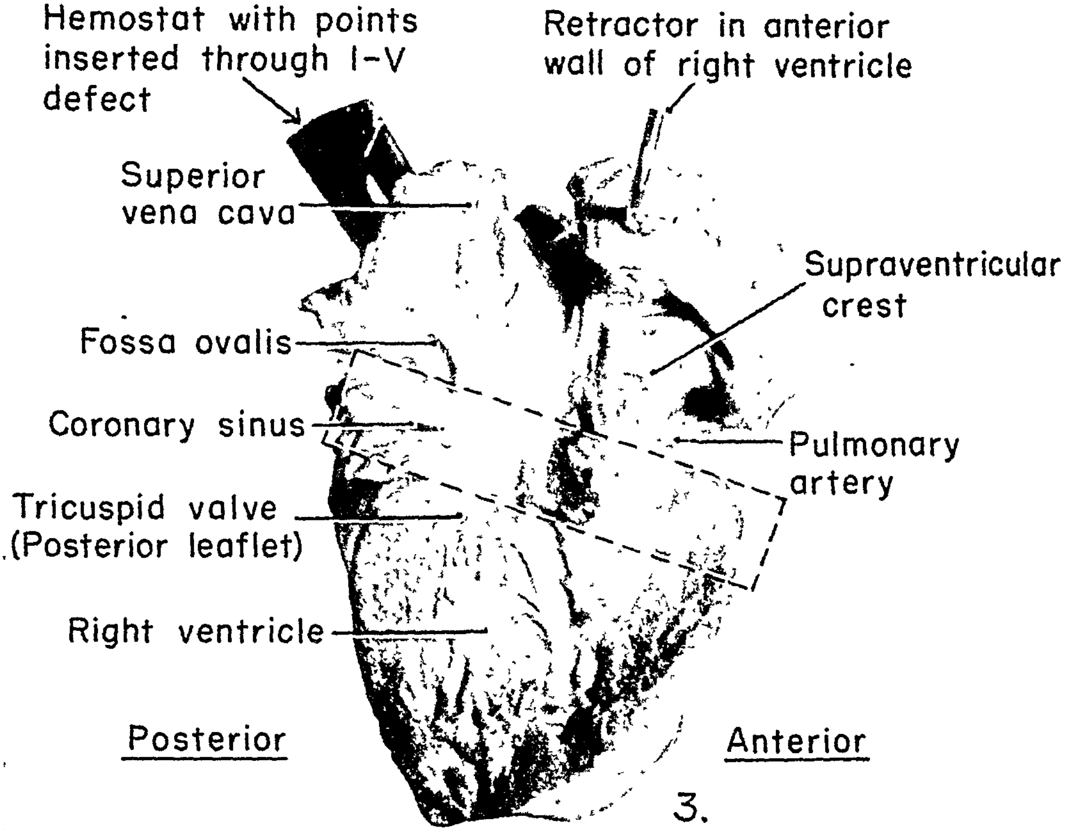
Hemostat with points inserted through I-V defect
Retractor in anterior wall of right ventricle
Superior vena cava
Supraventricular crest
Fossa ovalis
Coronary sinus
Pulmonary artery
Tricuspid valve (Posterior leaflet)
Right ventricle
Posterior
Anterior
3.

Fig. 3.—Interior view of right atrium and right ventricle of Specimen 2. The dotted rectangular area represents the region from which a wax model was reconstructed to demonstrate the location of the common bundle and its left and right branches. Compare with Fig. 7, *A*.

The microscopic appearance and relations of the common bundle are shown above the fibrous ring in Fig. 4, and as the bundle passed through this connective tissue layer at the atrioventricular junction in Fig. 5. The common bundle is identified below the fibrous ring as it lies in the free posterior margin of the interventricular septal defect (Fig. 5). Distally, the common bundle divided on the postero-inferior margin of the defect (Fig. 6) into a broad flat left bundle branch and a smaller right bundle branch. The relations of the microscopic right and left bundle branches to the defect (Fig. 6) are easily seen by comparing the microphotographs with the same structures in Fig. 7.

In view of the well-stained specialized muscle fibers, it was deemed desirable to demonstrate the crucial three dimensional relations of the conduction system in this specimen by means of a wax model reconstruction. Serial sections were projected and traced from the region of the atrioventricular node above, to the formation of the 2 bundle branches below. The region of the heart thus reconstructed by means of superimposed wax plates is indicated by the dotted rectangular area shown in Fig. 3.

fibrous ring at a lower level, to send fibers into the lower surface of the common bundle as well as into the top of the muscular interventricular septum posterior to the defect. A few muscle fibers were noted in the base of the posterior leaflet of the tricuspid valve in this specimen, but they had no relation to the myocardium of the interventricular septum or right ventricular wall.

Specimen 3. (No. A 549, white female, age 2 months.)

In addition to a high I-V defect, this specimen had a markedly dilated right atrium, patent foramen ovale, completely stenosed pulmonary artery, aorta overriding the I-V defect, and tremendous hypertrophy of both the left and right ventricular walls. The microscopic sections also demonstrated extensive hemorrhage within the atrioventricular node, common bundle and small left bundle branch.

This heart, like Specimens 6 and 8, demonstrated numerous variations of the conduction tissue. As shown in Fig. 8 this heart had an aberrant fascicle of specialized tissue that arose from the common bundle as it passed through the fibrous ring. This aberrant fascicle passed anteriorly above the defect and then descended in front of it. The remainder of the common bundle passed deep to what appeared to be a thickened crista supraventricularis and then coursed inferiorly in the posterior margin of the I-V defect. As the common bundle descended to the lower margin of the defect, it gave off a very small left bundle branch which traversed the top of the septum to reach the endocardium of the left side (Figs. 8 and 9). The small left bundle branch (Fig. 9) could be followed for only a short distance before it was lost within the muscle fibers of the septum due to hemorrhage and the poorly differentiated bundle tissue.

A small bundle of Kent was observed to connect the right atrium with the posterior portion of the interventricular septum. A separate bundle of fibers issued from the common bundle to descend and become lost within the septal myocardium posterior to the defect. The presence of these septal strands may account for the small size and composition of the left bundle branch in this specimen.

Specimen 4. (No. A 4752, Negro female, age 3 months.)

This heart had a transposition of the aorta as well as a very large anteriorly placed I-V septal defect. As demonstrated in Figs. 10 and 11 the transposed pulmonary artery was markedly dilated, whereas the aorta was of smaller diameter and possessed a thinner wall.

It will be noted in Figs. 10 and 11 that the common bundle, after crossing through the fibrous ring, descended posterior to the defect and was located under the endocardium on the left side of the heart. While in this left posterior position the common bundle divided into its left and right bundle branches at the point indicated by the black dot in Fig. 11. Both the left and right bundle branches were unusually small. The left bundle branch then continued distally as a broad but very thin band of fibers (Fig. 11). The smaller right bundle branch was followed as it traversed and descended from left to right within the myocardium of the upper interventricular septum (Fig. 10). A short distance below the lower margin of the defect the right bundle branch reached the right endocardial surface of the septum and gave off several rami.

The small size of the 2 bundle branches may be explained in part by the existence of 2 rather large accessory muscle bundles. The latter extended from the common bundle above the fibrous ring, to the cardiac musculature of the I-V septum posteriorly. The course of these 2 accessory pathways as they descended behind the tricuspid valve is shown by the arrows in Fig. 10.

Specimen 5. (No. A 5449, white male, age 5 months.)

This specimen also demonstrated a transposition of the great vessels, stenotic pulmonary artery, and a very large patent foramen ovale. The small left atrium appeared to be only an expanded terminus for the small pulmonary veins. A large high interventricular defect was placed anteriorly.

The close proximity of the common bundle to the postero-inferior margin of the septal defect is well shown by this reconstruction (Fig. 7, *A* and *B*).

In Fig. 7, *B* the origin and initial course of the left bundle branch can be observed as it passed distally under the septal endocardium of the left ventricle. Just below its origin, the left bundle branch divided into a large anterior division and a smaller posterior division. When the right ventricular surface is viewed (Fig. 7, *A*), the smaller right bundle branch can be observed as it courses anteriorly below the lower margin of the defect.

The dotted line in Fig. 7 indicates the location of the fibrous atrioventricular ring (annulus fibrosus). As illustrated in this model not all of the pale stained muscle fibers of the common bundle passed through the fibrous ring. A large mass of residual tissue remained on the right atrial side of the fibrous ring and formed the fingerlike ridge of tissue identified as an aberrant branch of conduction tissue in Fig. 7, *A*. From this aberrant ridge we were able to follow 2 slender twigs of pale stained fibers across the fibrous ring and into the posterior part of the interventricular septum. Distally, the remainder of this aberrant tissue broke up into smaller fascicles, and they in turn were lost within the connective tissue of the fibrous ring.

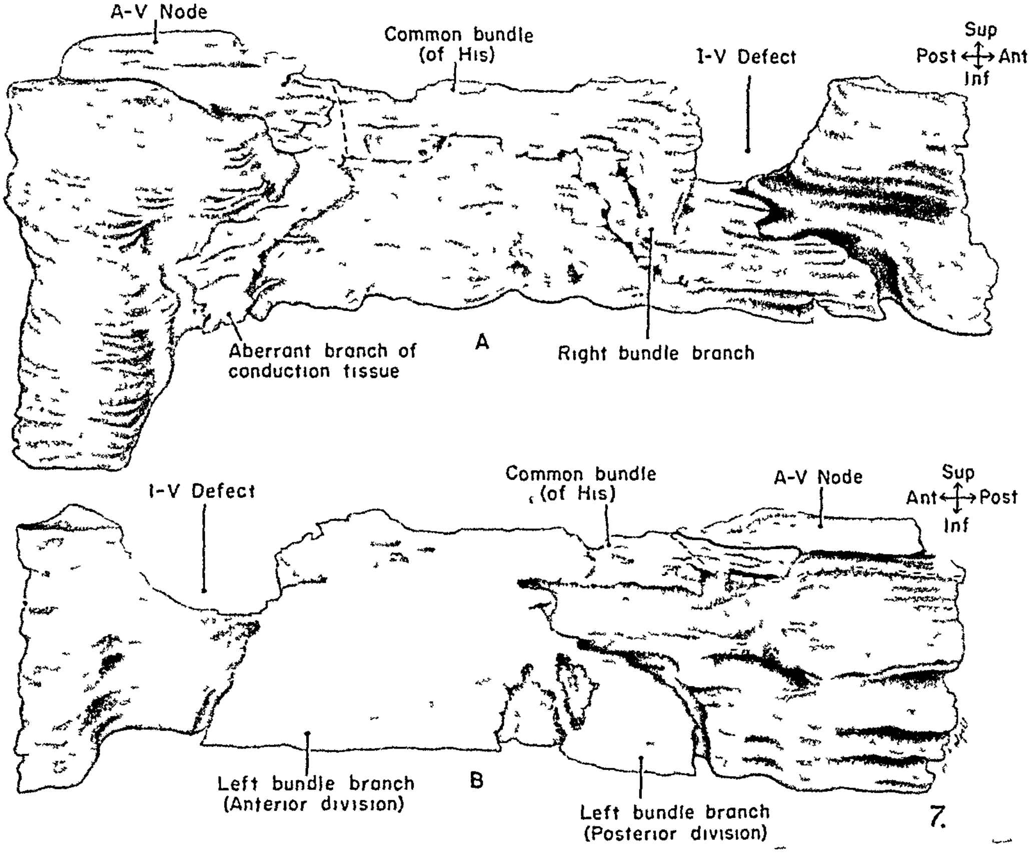

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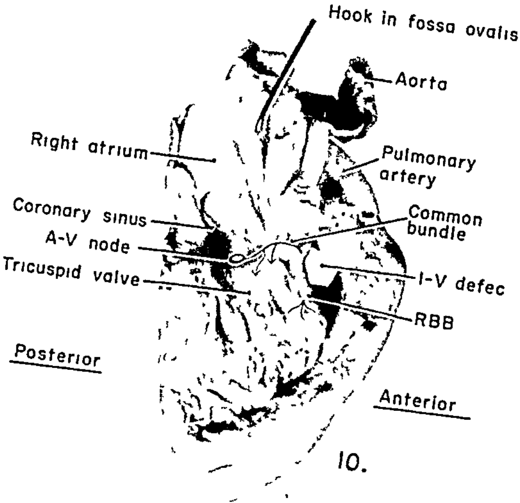
Fig. 7.—Drawing of a wax model reconstructed from the dotted area indicated in Fig. 3. The relations of the conduction tissue elements to the I-V defect are shown as viewed from the right ventricle in *A*, as viewed from the left ventricle in *B*. Note the position of the common bundle and its 2 branches to the postero-inferior margin of the defect. A large mass of pale stained fibers remained in the right atrium after the common bundle had passed through the fibrous ring (dotted line). Two small fascicles of fibers extended from this aberrant conduction tissue (Fig. 7, *A*) through the fibrous ring, and into the myocardium of the interventricular septum (i.e., bundles of Kent).

Not shown in Fig. 7 was a larger mass of such residual tissue that remained proximal to the fibrous ring nearer the atrioventricular node. This "left over" bundle tissue crossed the



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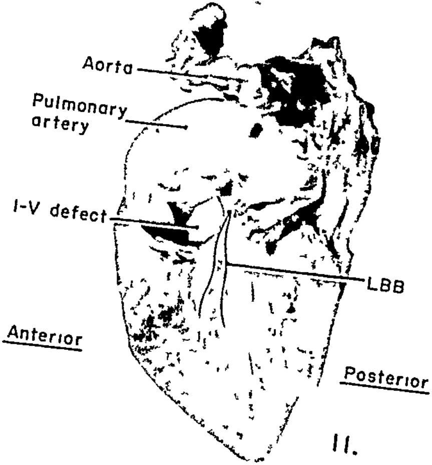

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Fig 10 —Interior of the right atrium and ventricle of Specimen 4 The intramyocard course of the right bundle branch (*RBB*) is dotted The arrows indicate the origin a course of 2 accessory pathways between atrium and ventricle (i e, bundles of Kent)

Fig 11 —Interior of left ventricle of Specimen 4 The common bundle divided (blac dot) on the left side of the heart and posterior to the I-V defect

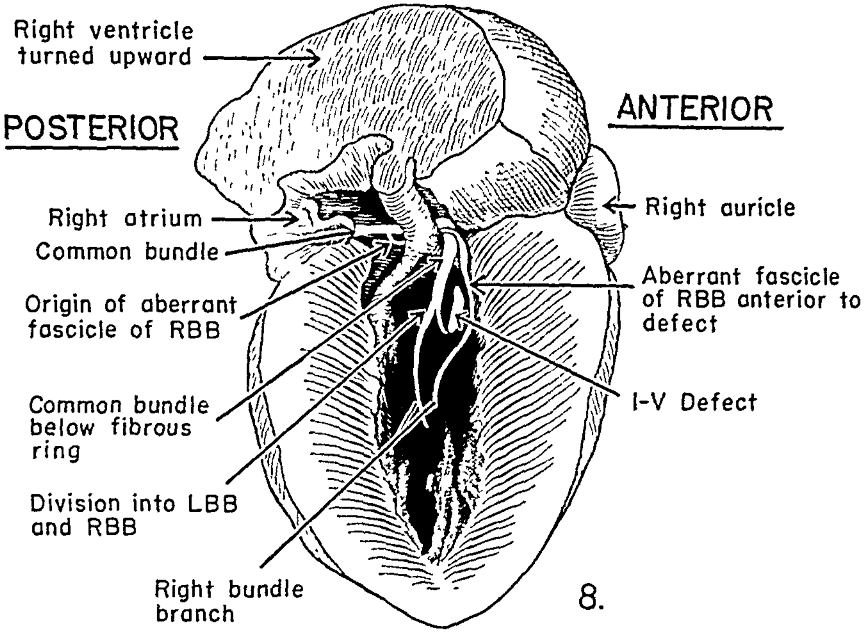

Right ventricle turned upward
POSTERIOR
ANTERIOR
Right atrium
Common bundle
Right auricle
Origin of aberrant fascicle of RBB
Aberrant fascicle of RBB anterior to defect
Common bundle below fibrous ring
I-V Defect
Division into LBB and RBB
Right bundle branch
8.

Fig. 8.—Drawing of the interior of the right atrium and ventricle of Specimen 3. The relations of the common bundle and its branches to the defect are indicated. Note the long slender aberrant fascicle of the right bundle branch (*RBB*) as it passed above and then descended anterior to the defect.

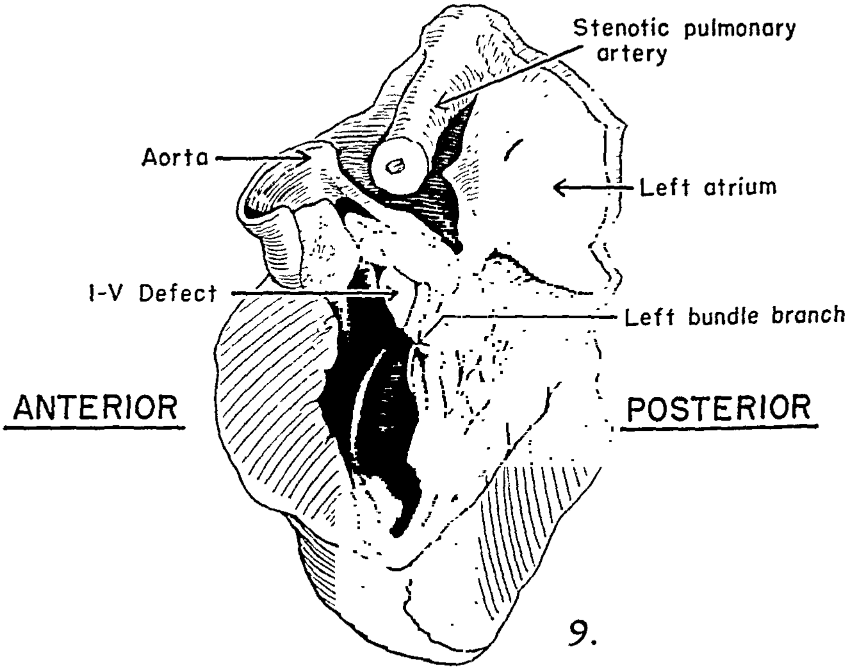


Fig. 9.—Drawing of the interior of the left ventricle of Specimen 3. The thin left bundle branch was extremely small in this heart (see text).

The left bundle branch descended posterior to the defect under the endocardium on the left side of the heart. It was located in the position normally occupied by the common bundle. Several rami were observed to leave the left bundle branch and enter the myocardium of the interventricular septum in the region of the defect.

The aberrant right bundle branch crossed the fibrous ring as 2 separate fascicles which entered the top of the septal myocardium. These 2 bundles never came to lie in the endocardium on the right side of the heart, but descended within the superficial part of the septal myocardium and were soon lost as discrete bundles. Although several pale stained fiber bundles in this specimen were traced into the fibrous ring, no accessory muscle bands were seen to cross the ring to connect the atrium above with the septum below.

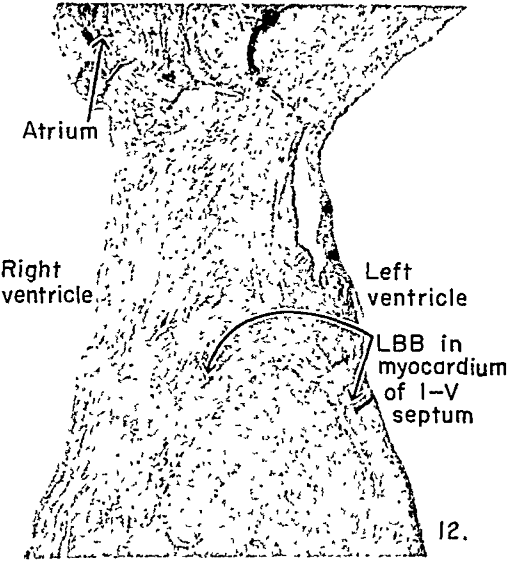


Fig. 12.—Microphotograph of a coronal section through the interventricular septum posterior to the 2 I-V defects of specimen 7. The common bundle divided under the endocardium on the right side of the septum. The left bundle branch (*LBB*) is shown as it coursed through the myocardium of the I-V septum to reach the endocardium on the left side of the heart. (Hematoxylin and eosin technique; ×40; reduced ⅓.)

Specimen 9. (No. A 54-51, white male, age 3 years.)

As illustrated in Figs. 13 and 14 this heart had a large high I-V defect, transposition of the great vessels, a dilated aorta that overrode the defect, and a stenosed pulmonary artery.

The common bundle again passed posterior to the defect, and after crossing the fibrous ring came to lie under the endocardium on the left side of the septum (Figs. 13 and 14). At the postero-inferior margins of the I-V defect the hemorrhagic common bundle divided (*x* in Fig. 14). As the left bundle branch continued distally, the right bundle branch coursed through the septal myocardium to gain the endocardium on the right side (Fig. 13). We again observed a strand of fibers leaving the common bundle as it divided. These fibers plunged immediately into the myocardium of the I-V septum posterior to the defect (arrow in Fig. 14).

The common bundle of conduction tissue in this specimen had a distribution quite similar to that shown in Figs. 1 and 2. It descended posterior to the septal defect, and divided into its 2 branches at the postero-inferior margin of the defect. The left bundle branch immediately assumed a flat, ribbon shape and gave off several rami to the upper portion of the septal myocardium as it descended in the endocardium on the left side of the heart. The small round right bundle branch was within the cardiac muscle as it descended on the right side of the septum (i.e., *deep to the endocardium*).

Although the distribution of the conduction tissue in this case was similar to that shown in Specimen 1, the present case had no rami that ascended anterior to the defect. However, as the common bundle divided into its 2 bundle branches, we could again trace several small fascicles from the lower end of the common bundle directly into the top of the interventricular septum.

Specimen 6. (No. A 6253, white male, age 6 months.)

This specimen had a large more inferiorly placed septal defect, transposition of the great vessels, and a patent foramen ovale.

There was a very small common bundle in this specimen. It was located under the endocardium on the left side of the septum. It descended and also divided into its terminal branches in the connective tissue above the septal defect. Both the left and right bundle branches were small, and both descended anterior to the septal defect near the junction of the anterior walls of the left and right ventricles. This is the only specimen studied microscopically in which both bundle branches descended anterior to a septal defect. An accessory bundle of Kent was also present in this specimen.

We observed numerous fascicles of large pale muscle fibers surrounding the transposed pulmonary artery. These fibers were similar to those found in the small common bundle. From this periarterial position we were able to follow this mass of pale fibers downward behind the defect into the endocardium of both the right and left sides of the interventricular septum. Below the I-V defect these fibers joined, and were incorporated with, the fibers of the left and right bundle branches. We were not able to establish histologic continuity proximally between the atrioventricular node and the mass of fibers observed around the transposed pulmonary artery. This was most unfortunate for we believe these periarterial pulmonary fibers were also a part of the conduction system and probably account for the small number of pale specialized fibers observed in the common bundle and the left and right bundle branches.

Specimen 7. (No. A 6256, Negro male, age 7 months.)

In addition to 2 I-V septal defects this specimen also demonstrated transposition of the great vessels, a patent foramen ovale, patent ductus arteriosus, and one large coronary artery that gave origin to the left and right coronary arteries.

The common bundle crossed the fibrous ring and descended posterior to both septal defects. As the common bundle descended it was located under the endocardium on the right side of the septum and it divided into its 2 bundle branches while in this position. At its origin from the common bundle, near the lower posterior margin of the defect, the left bundle branch then coursed through the upper myocardium to reach the left side of the septum, as shown in Fig. 12. The left bundle became a flat band of fibers as it descended and provided several rami that penetrated the musculature of the septum.

The smaller right bundle also gave origin to several early branches to the right side of the septum as it passed under the lower margin of the most posterior defect. A few small branches were seen to arise from the common bundle below the fibrous ring, prior to the origin of its 2 bundle branches. No accessory pathways were observed to connect the common bundle above the fibrous ring, with the interventricular septum below.

Specimen 8. (No. A 5466, white male, age 7 months.)

This heart demonstrated a small anteriorly placed I-V defect, transposition of the great vessels, and a patent foramen ovale. The common bundle divided into left and right bundle branches immediately above the fibrous ring in this heart.

Specimen 10 (No A 6254, white female [illegible]7 years)

In addition to 2 small IV defects, the heart had a very large interatrial septal defect

In this last specimen studied microscopically the common bundle again descended posterior to both IV defects. At the lower margin of the more posterior defect, the common bundle divided into [illegible] well differentiated but [illegible] branches. The left and right branches gave rami to the septal myocardium as they [illegible]ded in the endocardium of the left and right sides of the heart. No accessory muscles were observed to connect the atrial and ventricular myocardium in this specimen

Description of Dissected Specimens With the aid of a dissection microscope, the superficial layer of endocardium was partly removed to expose the regions of the atrioventricular node, common bundle and left and right bundle branches. Small pads of fat [illegible] connective tissue strands were removed in the area from the level of the coronary sinus above to the fibrous ring below. When viewed with moderate magnifications (i.e. ×5 to ×15), the fibers of the conduction tissue appear white and pale in contrast to the red of the regular cardiac muscle and yellow appearance of the fat. In formalin-fixed material the regular myocardial fibers are yellow to brown, whereas the conduction elements are white and del[illegible]. Engorged venous channels were also identified in the atrial regions of conduction tissue above the fibrous ring. However, the course and relation of the vascular channels were most conspicuous in fresh, unfixed specimens.

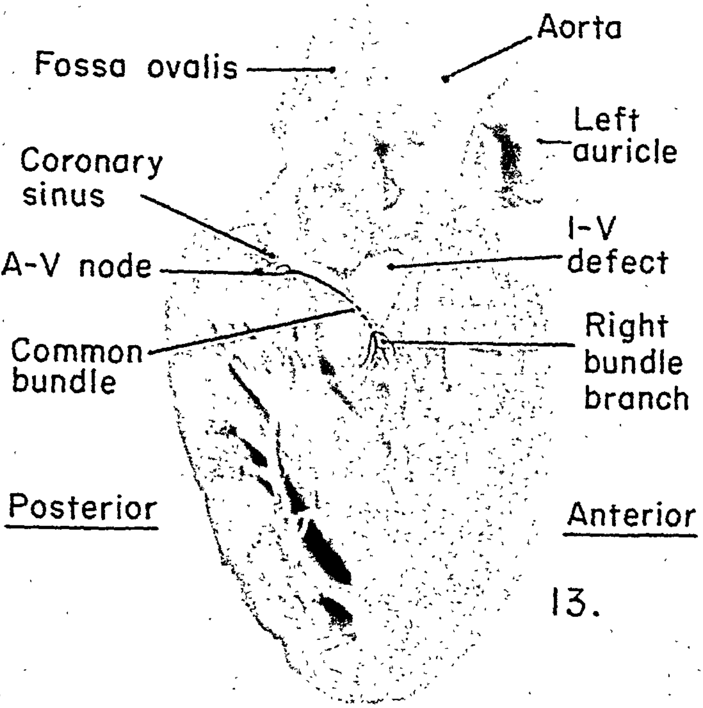
In 4 formalin fixed specimens (Specimen 13 excluded) we could identify the left bundle and its ramifications in the endocardium of the left ventricle with minimal dissection. For this reason the left bundle branch was exposed first, then the atrioventricular node, common bundle and right bundle branch. The latter was the most difficult structure to demonstrate due to its small caliber, acute angular course and early branching. A similar procedure was followed in each of the dissected specimens with highly satisfactory results.

Specimen 11 (No A 2082 white female, [illegible] days)

This heart possessed a high IV defect [illegible] The [illegible] was a discrete oval swelling located below the [illegible] the fibrous ring. The common bundle was [illegible] through the fibrous ring, and turned anter[illegible] to the defect. In this position it divided into [illegible] illustrations of Specimen 12 (see Figs 15 [illegible] posterior to the defect on the right side of [illegible] branch. The left bundle branch was a very [illegible] the defect. It reached and spread out over [illegible] 4 and 5 o'clock as the heart was viewed from a [illegible]

Specimen 12 (No A 5300 white male, age 5 [illegible])

In addition to a high septal defect [illegible] foramen ovale and hypertrophy of the left [illegible] dissection [illegible] the location of the defect [illegible] a small [illegible] in Fig 18, the atrioventricular node [illegible] of the [illegible] to the right along the upper [illegible] of the defect into [illegible] at the posterior margin of the defect [illegible] (EBB in Fig 18) descended [illegible] was soon lost in the endocardium



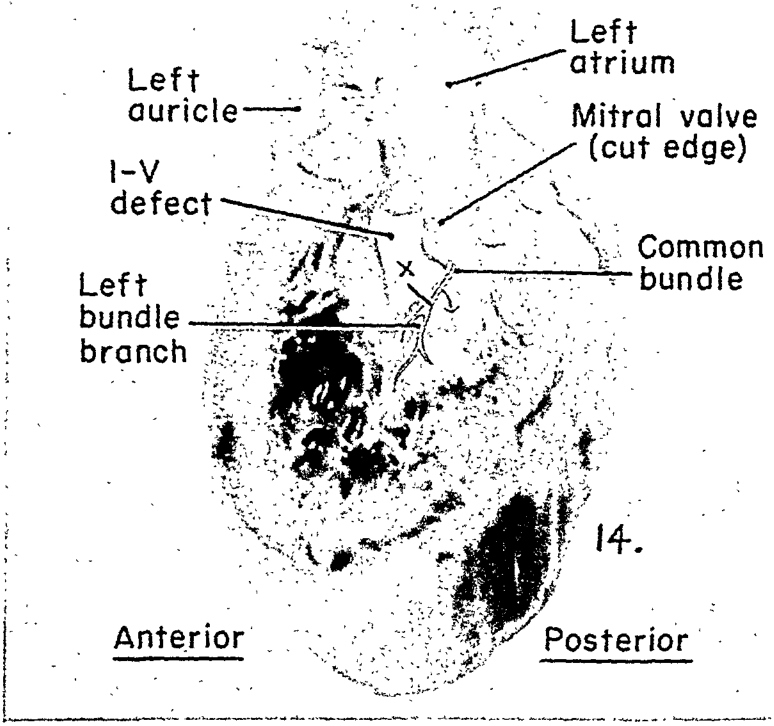


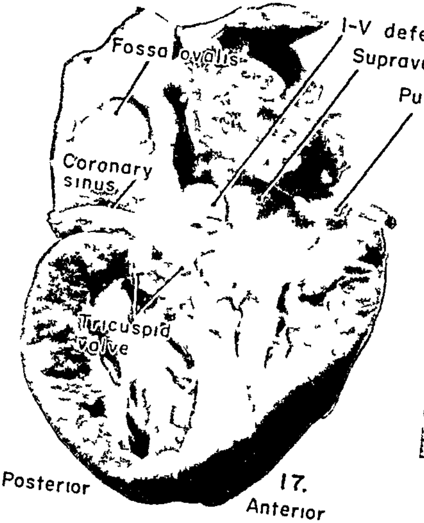
Fig. 13.—Interior of right atrium and ventricle of Specimen 9. The common bundle divided in the endocardium on the left side of the heart. Note the intramyocardial course of the right bundle branch (dotted) as it descended posterior to the defect.

Fig. 14.—Interior of left atrium and ventricle of Specimen 9. Note course of the common bundle as it passed deep to the cut edge of the mitral valve. The X marks the point of division of the common bundle into its 2 branches. The arrow indicates the origin and course of a fascicle of conduction fibers from the common bundle to the top of the interventricular septum (Mahaim's fibers).

divided almost immediately into its 2 well defined bundle branches at the lower margin of the small membranous portion of the I-V septum. The point of division was 10 mm. above the base of the papillary muscle of the conus and 8 mm. above and anterior to the small septal defect. Both the left and right bundles passed anterior to the defect as they descended on the left and right sides of the septum.

Specimen 15. (No. A 2-48, white female, age 10 months.)

A large defect 6 by 8 mm. was located posterior to the supraventricular crest, and opened into the right ventricle behind the medial cusp of the tricuspid valve. This heart demonstrated a patent foramen ovale and dilated aorta.



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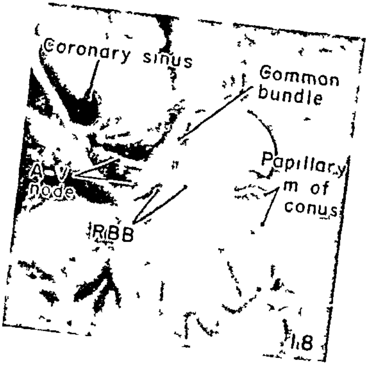

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Fig. 17.—Interior and topography of the right atrium and ventricle of Specimen 12.

Fig. 18.—Enlarged area of dissection to demonstrate the location and appearances of the atrioventricular node, common bundle and right bundle branch. Same specimen shown in Fig. 17. The remainder of the tricuspid valve and part of the endocardium were removed to expose the conduction elements.

A well defined atrioventricular node and common bundle were identified in the normal atrial position of this heart. The common bundle was traced through the fibrous ring to its division in the middle of the posterior margin of the defect. The origin and course of its 2 branches were very similar to those of Specimen 12 (see Figs. 17, 18). The right bundle branch was quite large, and descended in the free postero-inferior margin of the defect to reach the myocardium of the septum. Although the left bundle branch was only slightly larger than the right bundle, it assumed the shape and appearance similar to the bundle illustrated in Fig. 16.

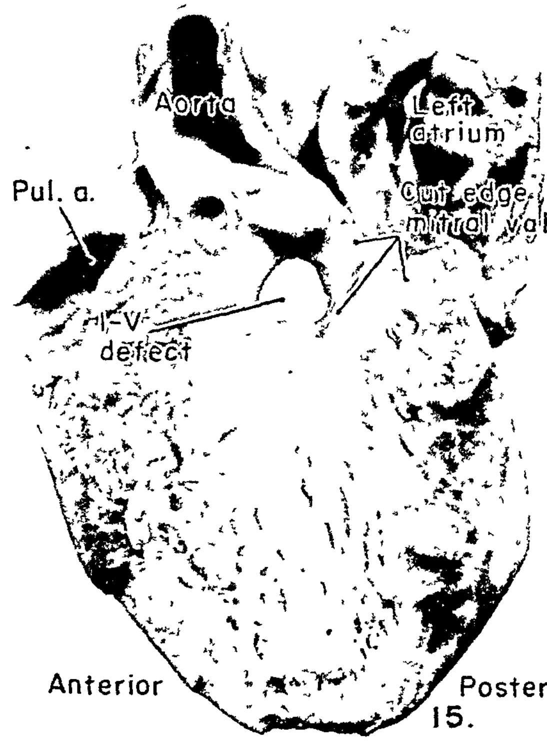
DISCUSSION

The results of the present investigation have demonstrated the position and branching of the common bundle (of His) along the postero-inferior margin of interventricular septal defects in 8 of the 10 human specimens

The left bundle branch (*LBB* in Fig. 16) was an ovoid funiculus at its origin, but rapidly broadened into a flat bundle that spread out superficial to the myocardium of the septum. Many smaller fascicles of conduction fibers could be observed through the endocardium as they coursed distally from the parent left bundle branch.

Specimen 13. (No. A 54 222, Negro female, age 3 months.)

This specimen was essentially a heart with a common atrioventricular canal. In addition to an atrioventricular defect that measured 12 by 9 mm., there was a patent ductus arteriosus.



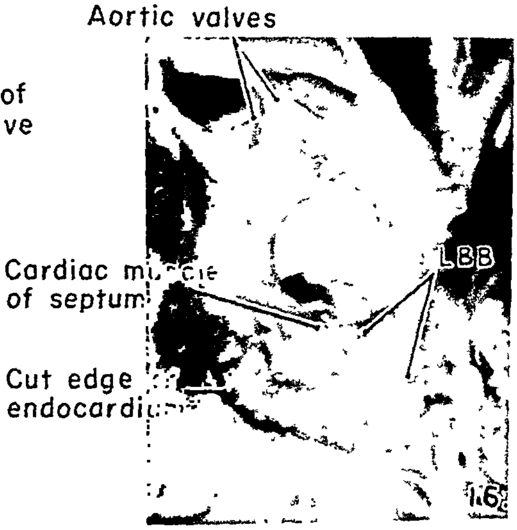


Fig. 15.—Interior of the left atrium and ventricle of Specimen 12. The I-V defect and topographic landmarks are identified.

Fig. 16.—Enlarged dissected area of left ventricle from the same specimen shown in Fig. 15. Note the gross course and position of the exposed left bundle branch (*LBB*) to the high I-V defect.

The atrioventricular node was a discrete ovoid mass. The common bundle passed through a tough mass of connective tissue as it coursed anteriorly to reach the posterior margin of the defect. Within this thick fibrous margin it gave origin to its branches.

The thin right bundle divided promptly into 2 smaller rami that descended on the right side of the septum. A small anterior ramus coursed anteriorly below the defect to attain the base of the papillary muscle of the conus, whereas a larger ramus descended more posteriorly on the septum. The thick left bundle branch plunged immediately into the myocardium at the top of the septum, and only a few fascicles could be identified in the endocardium on the left side of the septum.

Specimen 14. (No. A 9-53, white male, age 4 months.)

This was the only specimen that had a high septal defect located far *posteriorly*. The defect was only 2 mm. in diameter. This specimen also demonstrated a patent foramen ovale.

The atrioventricular node and common bundle were discrete structures and normally located within the right atrium. After passing through the fibrous ring, the common bundle observed microscopically in the hearts of patients who had manifested an antemortem Wolff-Parkinson-White syndrome by several recent investigators.[19-22] We observed no bundles of Kent in specimens that were beyond 6 months of age. It is obvious that such early branches of the common bundle, and the accessory pathways of Kent, become of paramount importance following surgical closure, particularly if the common bundle, or its left and right bundle branches, are injured or incarcerated by a ligature distally. Although one can not predict their presence, absence or number in a given heart, their anatomic identification here implies there might be a margin of safety in the event the common bundle or its branches were damaged distally.

It should be noted that the common bundle normally divided at the top of the I-V septum. In the present series this division occurred under the endocardium on the left side of the heart in 3 cases, and on the right endocardial surface in 2 other specimens.

Studies are now in progress in our laboratory on the degenerative and regenerative capacities of the conduction system. It is hoped that this work, as well as the physiologic approaches such as those of Scher,[23] and Scher and Young,[24] Draper and Weidemann[25] and Coleridge and his colleagues[26] will throw additional light upon the physiology of the conduction system. More extensive pathologic studies similar to those of Yater and associates[15, 16] and Lev and Unger[27] are also necessary.

The presence of hemorrhage within the A-V node and bundle branches of 6 specimens is difficult to explain inasmuch as no ante mortem arrhythmias were noted. It is possible that such hemorrhage occurred as a result of venous congestion and agonal spasm at the time of death. Thin walled veins and sinusoids from the myocardium are known to course through the conduction tissue,[28, 29] en route to the coronary sinus. These veins and sinusoids were engorged with blood in most of the specimens in the present series. One can also identify these vascular structures while dissecting above the fibrous ring in fresh unfixed hearts.

Final brief mention should be made concerning the location of the conduction system during the development of the heart. Walls,[11] in an excellent study of human embryos, has pointed out that these specialized fibers appeared to be related to the posterior wall of the atrioventricular canal. The posterior location of this specialized system in most of our anomalous older specimens would support Walls' belief. This was impressed upon us particularly as we exposed the incarcerated common bundle of Specimen 13. In this case the entire postero-inferior margin of the atrioventricular defect was a thickened tough, fibrous mass of connective tissue that encased the common bundle. We cannot say whether this represented a portion of the arrested remains of the dorsal endocardial cushion.

Further study of the development of the conduction system in younger human embryos is now being undertaken in this laboratory. We hope that the results of this newly initiated program will extend the observations of Walls,[11] and provide information for some of the present unanswered questions.

studied microscopically. It occupied a similar position in 4 of 5 hearts prepared under a dissecting microscope. This intracardiac relationship corroborated a preliminary demonstration,[10] and verifies the "presumed location of the conduction system" as illustrated by Kirklin and his co-workers.[6] Although all of the conduction tissue descended anterior to the defect in Specimen 6, and only a portion of it in Specimen 3, it should be recalled that additional fibers of the conduction system were present in both cases. In the former, we observed numerous aberrant fibers about the transposed pulmonary artery, while in Specimen 3 (see Figs. 8 and 9) the main portion of the common bundle and both bundle branches were posterior to the defect.

Thus, it would appear that these anteriorly placed conduction fibers would have been injured by ligatures, if a surgical attempt had been made to close the defects in these specimens by reported techniques.[4, 5, 8] However, it is doubtful that such damage would have caused a complete heart block in either case, in view of the more posteriorly placed branches of the conduction system.

It is worthy of special emphasis that Kirklin[6] has called attention to the presumed location of the conduction system in the inferior angle of the defect. He also noted that by protecting this area during recent surgical closure of septal defects in 25 patients, they had encountered no heart block. However, in an earlier series, 3 cases of heart block had resulted in 10 or 11 patients before they took the precaution of protecting the posterior and inferior margins of such septal defects. We believe the reason for the absence of heart block in Kirklin's revised surgical approach is explained by the anatomic findings reported in the present communication.

As one grossly examines such septal defects on the left side of the septum, as though looking at the face of a clock, the conduction tissue would appear to be most vulnerable to injury by ligature between 1 and 6 o'clock. When viewed through the right ventricle, this specialized muscle tissue would be most vulnerable to injury by ligature between 6 and 11 o'clock. To the best of the authors' knowledge no one knows just how much of the ventricular conduction system must remain intact to prevent complete heart block. One finds considerable speculation on this point, and the final answer must take into consideration the early rami that pass directly into the myocardium of the interventricular septum from the common bundle, prior to its division into left and right bundle branches. Such early branches from the common bundle after it passed through the fibrous ring were noted by Mahaim,[17] and called "paraspecific conduction fibers." In the present study, such fibers were traced from the common bundle into the very top of the I-V septum in 7 of the 10 specimens (e.g., arrow in Fig. 14). In addition, small fascicles were seen to leave the left bundle branch near its origin and enter the septum in 4 specimens.

We were surprised also to find accessory muscular pathways (bundles of Kent[18]) connecting the common bundle above the fibrous ring with the septal myocardium below the fibrous ring (Specimens 2, 3, 4, and 6). These aberrant bundles are in addition to the common bundle (of His). They were also

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SUMMARY

The position, course, and variations of the cardiac conduction system were studied in 15 human hearts with high interventricular septal defects. Ten specimens were studied by serial microscopic sections while the conduction elements were demonstrated grossly in 5 hearts with the aid of a dissecting microscope.

In 12 specimens the common bundle as well as the left and right bundle branches were located in the postero-inferior margin of the septal defect. These important relations of the conduction system are illustrated by means of photographs, drawings, and a wax model reconstruction. It is essential that the posterior margin of such high septal defects be protected from sutures and strong traction during surgical closure if complete or partial bundle branch blocks are to be avoided.

The *common bundle divided into its 2 bundle branches above the fibrous* ring in 1 specimen, and immediately above the septal defect in another (Specimens 8 and 6). The left and right bundle branches descended anterior to the defect in only 2 hearts (Specimens 6 and 14).

Accessory atrioventricular pathways of conduction tissue (bundles of Kent[18]) were demonstrated in 4 hearts. High branches from the common bundle (Mahaim's fibers[17]) into the top of the I-V septum were also noted in 7 of 10 specimens studied microscopically. The chief variations of the cardiac conduction tissue are presented as well as their physiologic and surgical significance.

Hemorrhage within the conduction tissue was observed in the microscopic sections of 6 hearts. Numerous, thin-walled, venous channels are known to traverse the conduction tissue en route to the coronary sinus and right atrium. Rupture of these endothelial channels during agonal spasm may account for the post-mortem hemorrhage and clinical absence of ante-mortem arrhythmia.

The authors express their sincere thanks to Drs. James B. Arey, Joseph E. Imbriglia, and William H. Miller for providing some of the specimens included in this study. We are indebted to Miss Marjorie Stodgell and Mr. Louis Sunny for the excellent medical art and photography used as illustrations in this communication. We are pleased to acknowledge the technical assistance of Martha Q. Smythe, Edna McCrane, Dr. Jean MacCreight, Irene Gamerman, and Anita Wilder.

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In congenital heart disease with impaired pulmonary circulation, as in tetralogy of Fallot, a rich subpleural network of small veins in the hilus is formed. This venous network connects the pericardiophrenic and parietal venous systems with the pulmonary circulation forming a collateral circulation. It seems logical that these vessels transport venous blood which bypasses the right heart and flows directly into the lungs.[4] This clinical observation gave us the idea to elaborate the procedure by surgical anastomosis.

EXPERIMENTAL OBSERVATIONS

Mongrel dogs of both sexes were used. The operations were performed under endotracheal anesthesia. The chest was opened in the right third intercostal space anterolaterally. The pleura covering the superior vena cava was incised longitudinally and the vein freed in its entire length. The main branch of the right pulmonary artery was transsected and the peripheral stump was held in an atraumatic clamp. The superior vena cava was then ligated just superior to the azygos vein. Between the ligation of a second clamp, the vena cava was severed. The peripheral stumps of the transsected superior vena cava and the right main branch of the pulmonary artery were approximated by an end-to-end anastomosis. Upon removing the clamps, the right pulmonary artery readily filled from the superior vena cava. The azygos vein was ligated last and the chest was closed layer by layer (Fig. 1).

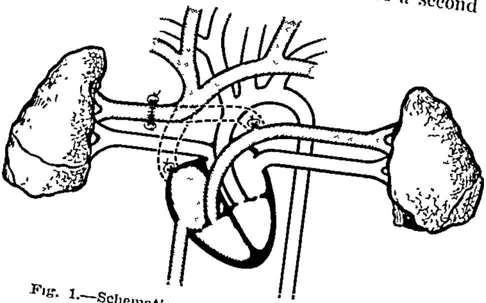

Fig. 1.—Schematic representation of the method.

As a result of this operation, the venous blood flow is divided into two parts: the blood of the upper part of the body, which bypasses the right heart, flows from the superior vena cava directly into the right lung; the blood of the lower body flows ''per vias naturales'' through the right heart into the left lung (Fig. 2).

At 3- to 12-month intervals following the operation, the respiratory function and the hemodynamics were studied in the following manner.

The oxygen uptake was determined separately for the left and for the right lung by selective bronchospirometry. At the same time, an intravascular catheter was introduced into the right external jugular vein

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VENA CAVA—PULMONARY ARTERY ANASTOMOSIS FOR VASCULARIZATION OF THE LUNG

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CHARLOTTE, N. C.

ONE of the most difficult and most interesting problem in modern cardiology is the surgical treatment of complex cardiac malformations in the "impaired pulmonary circulation" group. A survey relevant to this problem shows that despite the remarkable results obtained there is still much to be desired. The surgical management for certain pathologic cardiac conditions, such as the noncomplicated pulmonary stenosis, has been solved theoretically and technically. The direct vision repair of tetralogy of Fallot under certain conditions has become a reality. On the other hand, conditions, such as tricuspid atresia, still await effective therapy, and the same applies to certain types of tetralogy of Fallot when the stenosis involves the entire length of the right ventricular outflow tract or when the pulmonary artery is excessively hypoplastic or atretic.

The Blalock[1] operation, or its modification developed by Potts, Smith and Gibson,[2] restores function satisfactorily in numerous cases, but has the following disadvantages.

1. The "patent ductus" created artificially increases greatly the left ventricular output and places considerable strain on the heart.

2. In the presence of high-grade pulmonary stenosis and relatively slight aortic dextroposition with small interventricular septal defect, the stress upon the right ventricle is great because the right ventricle has to force blood through very small apertures in the direction of both great vessels. A subclavian–pulmonary or aorta–pulmonary anastomosis may result in an increase of pulmonary arterial pressure, which in turn makes it even more difficult for the right ventricle to express the blood through the narrowed pulmonary orifice. Thus, right ventricular failure may develop.

3. The blood supplied to the lungs by way of the anastomosis is mixed arterial and not venous; therefore, the efficiency of the anastomosis is reduced.

4. There is a tendency to endarteritis.

5. The technique is exceptionally complicated especially with children.

The above considerations caused us to look for a surgical method[3] which would eliminate the disadvantages outlined.

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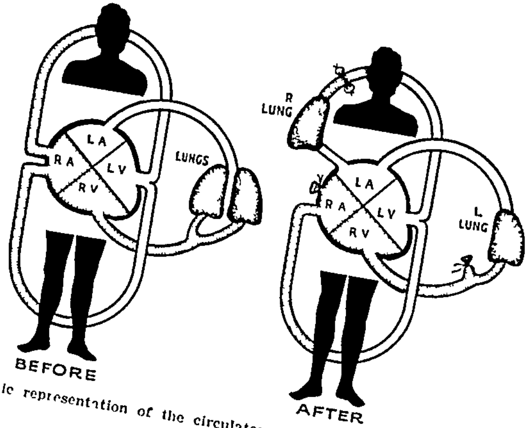

Fig. 2.—Schematic representation of the circulatory relations before and after the operation.

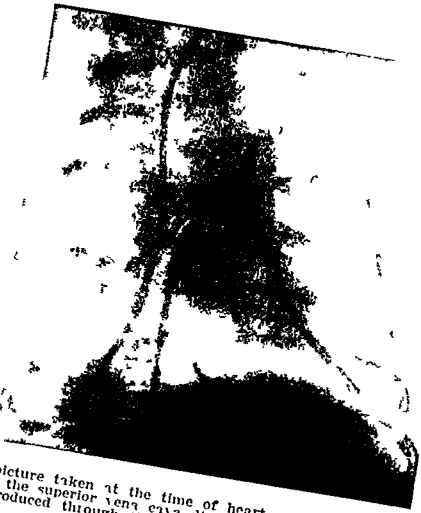

Fig. 3.—X-ray picture taken at the time of heart catheterization. The upper catheter is introduced through the superior vena cava directly into the right pulmonary artery. The lower catheter is introduced through the vena cava inferior and right heart into the left pulmonary artery.

into the right pulmonary artery via the anastomosed superior vena cava. Another catheter was passed through the femoral vein into the inferior vena cava and then through the right heart into the left pulmonary artery (Fig. 3). Samples of blood taken from the catheters and by direct arterial puncture were analyzed for oxygen content by photometry. Detailed records were made of blood pressure.

The blood flow through the right and left lungs, respectively, was calculated on the basis of the Fick principle, according to the following equations.

$$F_{ll} = \frac{O_r}{C_{art} - C_{rpa}}.100 \tag{1}$$

$$F_{rl} = \frac{O_l}{C_{art} - C_{lap}}.100 \tag{2}$$

Pulmonary vascular resistances in the right and left lungs were calculated according to the following equations.

$$PVR_r = \frac{MP_{rpa} - MP_{cap}}{\left(\frac{F_{rl}}{60}\right)}.1 \times 332 \tag{3}$$

$$PVR_l = \frac{MP_{lpa} - MP_{cap}}{\left(\frac{F_{ll}}{60}\right)}.1 \times 332 \tag{4}$$

where

F_{rl} = right lung blood flow in ml./min.
F_{ll} = left lung blood flow in ml./min.
O_r = oxygen uptake of the right lung in ml./min.
O_l = oxygen uptake of the left lung in ml./min.
C_{art} = blood oxygen content in the systemic arteries in vol. %.
C_{rpa} = blood oxygen content in the right pulmonary artery in vol. %.
C_{lpa} = blood oxygen content in the left pulmonary artery in vol. %.
MP_{rpa} = mean blood pressure in the right pulmonary artery in mm. Hg.
MP_{lpa} = mean blood pressure in the left pulmonary artery in mm. Hg.
MP_{cap} = pulmonary "capillary" mean blood pressure in mm. Hg.
PVR_r = "pulmonary vascular" circulatory resistance in the right lung in dyn. sec. cm.$^{-5}$.
PVR_l = "pulmonary vascular" circulatory resistance in the left lung in dyn. sec. cm.$^{-5}$.
1.332 = conversion factor.

Angiocardiographic examinations were carried out by means of an intravascular catheter introduced into the superior vena cava. Thirty milliliters of a 70 per cent preparation of Joduron were injected through the catheter into the superior vena cava, and the passage of the contrast medium was followed by serial roentgenography.

Finally, the circulatory effect of (a) obstruction of the left main bronchus, and (b) obstruction of the left pulmonary blood flow were studied.

The purpose of these experiments was to control the function of the right lung anastomosed with the superior vena cava in the absence of the function of the normal (left) lung. These experiments were performed with the aid of double lumen catheters provided with inflatable cuffs introduced into the left main bronchus (series No. 1) and into the inferior vena cava above the level of the diaphragm (series No. 2).

normal. *Half of this group was sacrificed 6 months after operation, the others* were allowed to survive for further observation and examination.

At necropsy, the veins of the superior caval system were questionably larger. This was the only sign suggesting venous congestion. The vessels at the anastomotic site were patent and the suture line was completely covered by endothelium. There were no pathologic changes in the lungs histologically.

Bronchospirometry and hemodynamic studies were performed on 10 dogs. Relevant data on the respiratory function and the results of hemodynamic tests are summarized in Table I and Figs. 4, 5, 6, and 7.

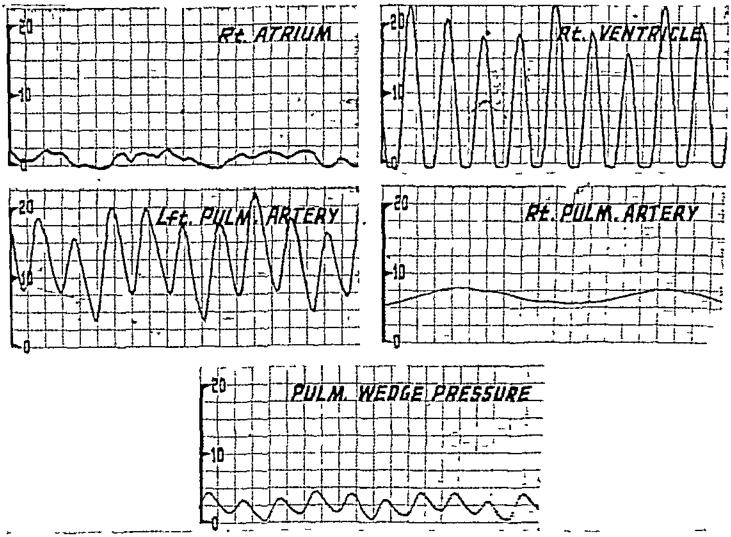

Fig. 6.—Postoperative pressure curves.

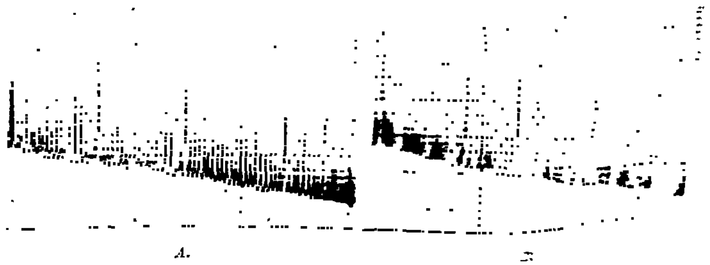

Fig. 7.—Postoperative [illegible]

RESULTS

End-to-end anastomoses between the superior vena cava and right pulmonary artery were performed in 30 dogs. Six dogs were lost in consequence of the operation. One died of empyema, one of heart fibrillation, 2 from obliteration of the anastomosis.

Twenty-two of the 24 survivors had patent anastomoses 3 months to 2 years after operation. One to 2 weeks after operation the behavior of the animals was

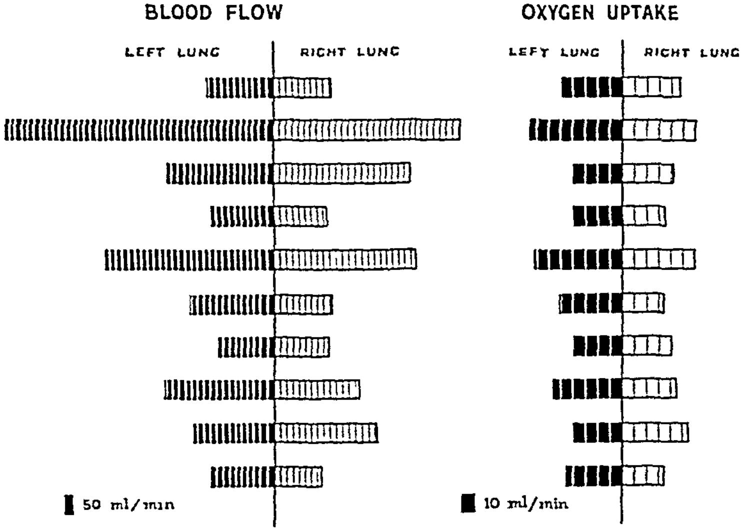

Fig. 4.—Pulmonary blood flow and oxygen uptake postoperatively of the right and left lungs.

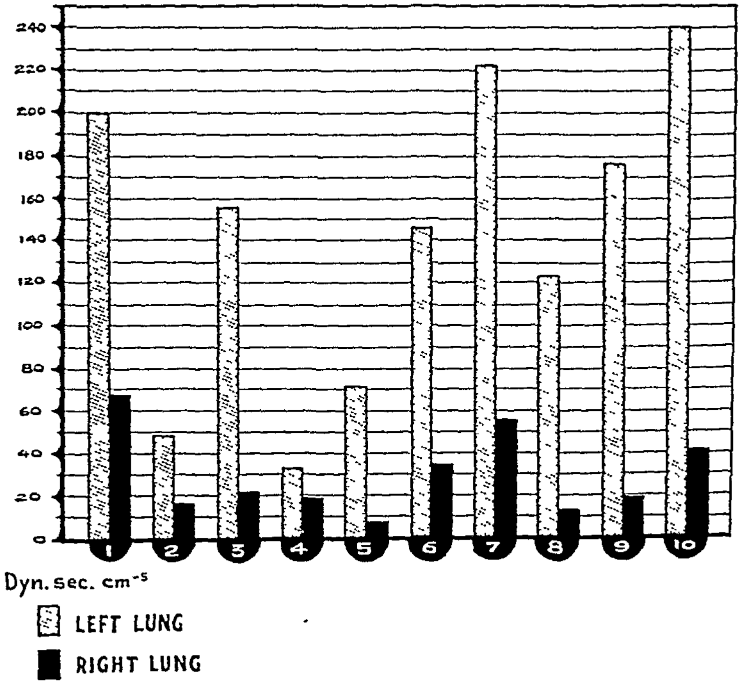

Fig. 5.—Pulmonary "vascular" circulatory resistance after operation.

The air passage of the left lung was obstructed in 5 dogs for 15 minutes, and the blood flowing into the left lung was also occluded for the same length of time. All dogs survived the 15 minutes of occlusion. The significant hemodynamic alterations in the group of the obstructed bronchus were slight elevation of the systemic arterial blood pressure, marked hypertension in the left lung and oxygen desaturation of the systemic arterial blood (Figs. 9 and 10).

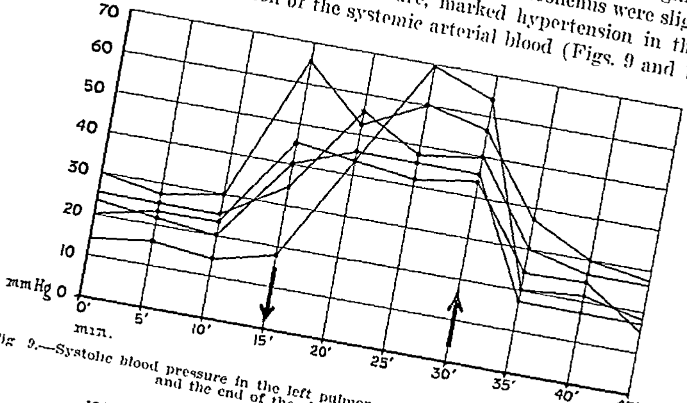

Fig 9.—Systolic blood pressure in the left pulmonary artery. The arrows mark the beginnin and the end of the obstruction of the left bronchus

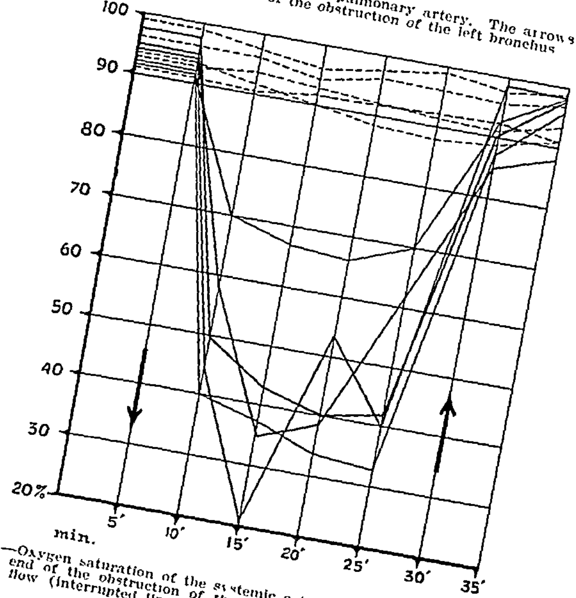

Fig. 10.—Oxygen saturation of the systemic arterial blood. The arrows mark the begin- and the end of the obstruction of the left bronchus (continuous line) or the left pul- ry blood flow (interrupted line).

Angiocardiographic examinations were performed on 6 animals. Serial roentgenography clearly showed the contrast material introduced into the superior vena cava filling the right pulmonary artery with its tributaries immediately (Fig. 8). The photograph taken one second later reveals normal parenchymal filling of the right lung. Two seconds later no contrast medium was detectable in the superior vena cava; the left heart and the aorta showed intensive concentration of the dye

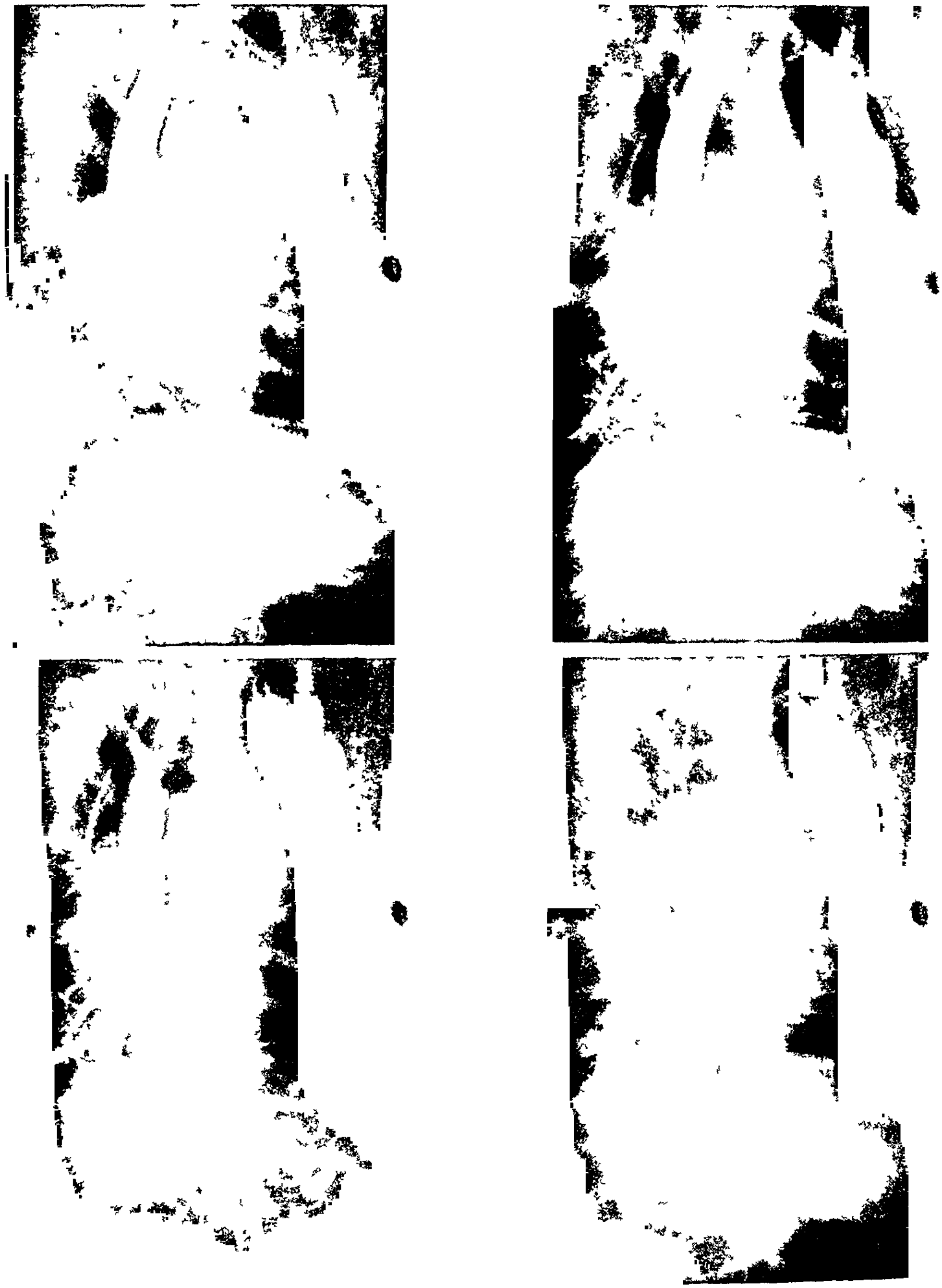

Fig. 8.—Postoperative angiocardiography.

resistance was considerably lower at the site of the anastomosis than in the contralateral lung (see Fig. 5). It is believed that an active dilatation of pulmonary vessels (primarily in the arterioles) is the result of a reflex process.[9]

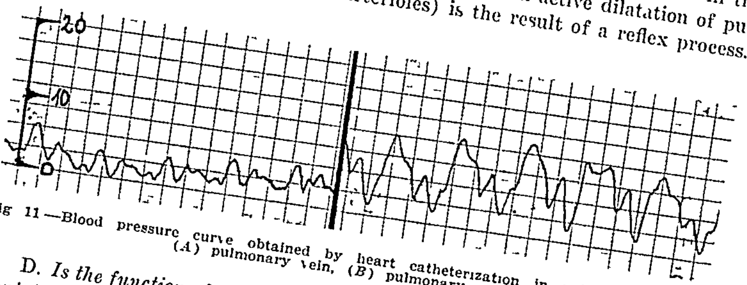

Fig 11—Blood pressure curve obtained by heart catheterization in pulmonary stenosis, (A) pulmonary vein, (B) pulmonary artery

D. *Is the function of the lung attached to the systemic venous system equivalent to that of a normal lung?:* Hemodynamic studies showed nearly equal blood flow, and bronchospirometry revealed nearly equal oxygen uptake of both lungs (see Figs. 4-7). The right lung attached to the superior vena cava proved to be functioning satisfactorily. To check this function further biologically, we excluded the left lung from the respiratory process. Two different methods were used.

The first method was to prevent air getting into the left lung. We found that the obstruction of the left bronchus was followed by severe circulatory changes, i.e., pulmonary hypertension, significant cyanosis and desaturation of the systemic arterial blood (see Figs. 9 and 10). When the right lung is functioning normally, obstruction of the left bronchus does not produce such symptoms. On the first look we must suppose that the function of the right (anastomosed) lung of our experimental animals was impaired, but when we consider the specialties of the pulmonary circulation of our animals we must accept other suppositions. In the case of normal pulmonary circulation, when we obstruct the main bronchus of one lung, the blood flow of this lung suddenly diminishes because the rise in circulatory resistance directs the blood to the other lung.[10] It is the same situation in unilateral pneumothorax. However, when pulmonary circulation is divided into two separate parts, such is not possible. In spite of the reflex pulmonary vasoconstriction, which accounts for the hypertension in the left lung, the blood flowing from the inferior vena cava *must* flow through the left lung, as there is no other route. The desaturation of the systemic arterial blood and the cyanosis also must be seen as a consequence of a great veno-arterial shunt through the nonoxygenated left lung and not as a consequence of an impaired function of the right (anastomosed) lung.

This supposition was supported by a second group of experiments in which the function of the left lung was impeded by obstruction of the left pulmonary blood flow. It was found that, in spite of the fact that only the right lung was functioning, no signs of cyanosis or systemic arterial desaturation occurred (see Fig. 10).

In the group of obstructed left pulmonary blood flow, the only significant alteration was the decrease of the systemic arterial blood pressure. Oxygen desaturation of the systemic arterial blood did not develop.

TABLE I

NO.	BLOOD PRESSURES MM. Hg		
		PULMONARY ARTERY	
	VENA CAVA SUPERIOR	RIGHT	LEFT
1	7/2 (5)*	7/2 (5)	30/7 (15)
2	9/7 (8)	9/7 (8)	28/8 (18)
3	10/3 (7)	9/3 (6)	32/16 (20)
4	8/6 (7)	8/6 (7)	20/4 (8)
5	6/1 (4)	6/1 (4)	24/10 (14)
6	7/5 (6)	7/5 (6)	25/9 (16)
7	7/4 (6)	7/4 (6)	35/7 (15)
8	6/2 (5)	6/2 (5)	25/10 (14)
9	7/2 (5)	6/2 (4)	26/12 (16)
10	6/3 (5)	6/3 (5)	26/16 (18)

*Mean pressure.

DISCUSSION

In evaluating the results of our investigations and the efficiency of the method, the following points were considered.

A. *What are the technical difficulties of the method?:* Experience has shown that the operation does not involve any major difficulties technically. In the Blalock and Potts operations, the anastomoses are usually under tension. In the operation under discussion, the stumps of the two vessels are brought together with ease. This method can be carried out by any surgeon practiced in suturing blood vessels.

B. *What are the effects upon the pressure relations of the superior caval system?:* Although average blood pressure in the superior vena cava was a few mm. Hg higher than normal, this increase of pressure was not significant and caused no untoward symptoms. Two of the early experimental animals died in the first postoperative weeks as a consequence of the thrombosis at the anastomotic site. Excessive venous congestion and edema of the upper part of the body developed in these 2 dogs.

C. *Can the systemic venous pressure provide adequate blood flow through the lung?:* The above hemodynamic problem is one that ultimately decides whether or not the operation is practicable. It is known that the blood pressure is relatively low in the pulmonary circulation even in normal conditions and the pressure difference between the pulmonary artery and vein is no higher that 5 to 10 mm. Hg.[5, 6]

In conditions associated with pulmonary stenosis, the mean pulmonary arterial pressure is even lower (4 to 8 mm. Hg) and in spite of the small pressure difference between pulmonary artery and vein, the blood flow in cases of noncomplicated pulmonary stenosis does not decrease below normal values (Fig. 11). The cause of this may be the high adaptability of the vessels of the lungs.[7, 8] In support of this view, our experiments showed that pulmonary vascular

The authors are indebted to Mrs Ruth Crooks for valuable technical assistance

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E. *What is the effect of this method on the burden of the right ventricle?:* In congenital malformations associated with pulmonary stenosis, the narrowing of the pulmonary orifice places a great extra burden on the right ventricle. The work of the right ventricle can be expressed by the following equation:

$$W = Q.R\ \frac{mv^2}{2g}$$

where (W) is the work of the ventricle, (Q) represents the systolic discharge, (R) the mean pressure or resistance, (m) the mass of the ejected blood, and (v) the average velocity.[11] Because of the relative quantity, the $\frac{mv^2}{2g}$ is negligible.

The anastomosis created by the Blalock or Potts operations does not diminish the burden of the right ventricle. Contrarily in some cases, there is an elevation of the factor (R) pressure in the pulmonary artery. This increases the burden upon the right ventricular myocardium. The method being reported has no such effect. There is no elevation in the factor (R), but there is a great diminution of the systolic discharge (Q) of the right ventricle because the blood of the upper part of the body is shunted directly into the right lung.

F. *Is the method adaptable to the human being?:* At first glance, the operation described appears to be too hazardous to be recommended for use in human subjects. Nevertheless, it should be mentioned in connection with this problem that several operations now in use for the surgical treatment of congenital heart disease appeared to be fairly risky and complicated at first. Observations and experience in thoracic surgery show that the temporary clamping of one of the main branches of the pulmonary artery or the superior caval vein has no grave consequences even for the patient who is in poor condition.[12, 13]

SUMMARY

The authors developed experimentally a new method for the relief of certain congenital heart diseases associated with impaired pulmonary circulation. The essence of the procedure was as follows: the superior vena cava was transsected before entering the right atrium; the right pulmonary artery was severed at its origin with the main pulmonary trunk; the peripheral stumps of the two vessels were anastomosed by an end-to-end method. The right lung was thus supplied with blood directly from the superior vena cava. In this manner about half of the sytemic venous blood flow bypassed the right heart.

Animal experiments have shown that pulmonary blood flow and oxygen uptake are nearly equal on both sides. There is no significant increase in blood pressure in the area of the superior vena cava. The method as illustrated has advantages over other anastomotic operations in the following respects: (1) the operation places no extra burden on the heart; (2) blood pressure in the main trunk of the pulmonary artery is not increased; (3) this anastomosis supplies "pure" venous blood to the lung; (4) endarteritis is less likely to occur; and (5) technically the operation, even on small subjects, is not difficult.

Detailed physiologic changes, hemodynamics, and angiocardiography studies are reported.

because of local unresectability or metastases. This procedure is neither innocuous nor inexpensive and may result in postoperative morbidity or mortality. Exploration without resection produces needless discomfort. The patient deteriorating with extensive cancer rarely returns to preoperative level when he survives. Churchill and his co-workers[2] report a mortality rate of 10.6 per cent for exploratory thoracotomy in their series of cancer of the lung from 1933 to 1950. This figure has dropped considerably of late and is probably less than 2 per cent today. It has been with the hope of avoiding fruitless thoracotomies that this study has been undertaken.

The investigation has been concerned with the value angiocardiography may have as an aid to the recognition of certain nonresectable lung carcinomas. It had seemed probable that in centrally located tumors, especially, this procedure would be most useful. Even in the peripherally placed ones it may occasionally offer informative data concerning resectability by disclosing changes in the mediastinum or hilar structures. It has been our primary aim to determine the degree of accuracy of this prognostic measure.

The findings in a group of 23 individuals, all of whom had been submitted to preoperative angiocardiography, were checked at surgical operation. One of us (E. W. W.) was in attendance at 15 of the operations; detailed operative notes were available for review in all cases.

METHOD USED IN ANGIOCARDIOGRAPHY

Examinations were performed initially using the original method of Robb and Steinberg, namely a No. 12 cannula inserted into and tied in one of the deep arm veins through which was introduced a concentrated iodine compound. At first, 50 per cent Diodrast was used, but this has been replaced by 70 per cent Urokon. Serial films were made, originally by a homemade cassette-changing device which permitted 20 exposures at rates up to 3 per second, in one projection. More recently a biplane roll film changing device (Elema) has been employed providing simultaneous anteroposterior and lateral recording at rates up to 12 frames per second in each projection. Most examinations had been done at the rate of 6 frames per second for 2 or 3 seconds followed by 2 per second for an additional 5 or 6 seconds, and finally 1 per second for a total run of 15 to 20 seconds. The method of injection has also been changed, as experience dictated, using a No. 11 or 12 Lehman catheter introduced via the deep veins of the arm on the affected side into the corresponding innominate vein, and the compound injected by means of a central-bore Robb hand-operated syringe. The method has been quite satisfactory, permitting the introduction of between 50 c.c., and at the most 70 c.c., of 70 per cent Urokon in less than 2 seconds. This part of the procedure has been performed by the surgeon with the radiologist determining the proper positioning of the patient in addition to other radiographic factors. General anesthesia has not been needed or used although an anesthesiologist has attended each examination.

Twenty-four examinations were carried out in these 23 patients without serious incident. (Patients with evidence of cyanosis, recent myocardial infarction, asthma or serious allergy have not been accepted for this examination.) Two patients had febrile responses, one to 104° F., within 4 hours but

ANGIOCARDIOGRAPHY AS AN AID TO IDENTIFICATION OF NONRESECTABLE PULMONARY CARCINOMAS

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DEVELOPMENT OF ANGIOCARDIOGRAPHY AND ITS DIAGNOSTIC POTENTIALITIES

IN THE 18 YEARS since angiocardiography was first established as a clinical method by Robb and Steinberg,[5] much valuable work has been done in the evaluation of cardiovascular abnormalities, lesions of the mediastinum, and various forms of pulmonary pathology. Carcinoma of the lung has been one of the most recent major subjects to be so investigated, and the present discussion will deal with the accuracy of preoperative determination of its resectability.

The diagnosis of pulmonary carcinoma, and the role that angiocardiography may play in it will not be considered at this time. A number of writers[4] have devoted much effort to such studies and believe that angiocardiography plays a vital part in both the diagnosis and determination of resectability. The studies carried out at the Massachusetts General Hospital on the other hand confirm the opinion of Dotter, Steinberg, and Holman[3] and Amundsen and Sörensen[1] that, by angiocardiography, pulmonary carcinoma may be indistinguishable from certain other conditions and that its diagnosis must be established otherwise.

PREDICTION OF RESECTABILITY OF LUNG CARCINOMA

Certain clinical signs suggest that total resection may not be possible. These include phrenic or recurrent nerve paralysis, Horner's syndrome, cytologically positive pleural effusion, and distant metastases. Evidence of hilar or mediastinal extension, however, may not be apparent by conventional examination, either clinical or roentgenologic. Complete extirpation of carcinoma of the lung is impossible when it or its local metastases lie in intimate contact with vital intrathoracic structures which cannot be sacrificed. Principally these structures are the heart and great vessels. Involvement of the main right and left branches of the pulmonary artery, too close to the main trunk to permit resection, precludes removal of the tumor. Tumor invasion of the pulmonary veins close to the left atrium, or involvement by cancer of the superior vena cava prevents removal unless elaborate substitution of vessels is performed. Tumor lying in apposition with the aorta or great arteries to the head and arms may be resectable unless there be true invasion of these structures.

It is well known that most individuals with primary carcinoma of the lung are incurable when first seen. The figure varies with different authors but may run as high as 75 per cent. By presently accepted procedure, all patients are subjected to exploratory thoracotomy unless very obviously incurable

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13	912178	1 Obstruction of rt innominate vein and vena cava	Not resectable	Not resectable	Extension of tumor from RUL to great vessels and trachea
14	240628	Mass lying close to descending thoracic aorta	Resectable	Not resectable	Tumor invading aorta and posterior chest wall
15	956091	*Left injection* Extrinsic obstruction of lt innominate vein, by innominate artery *Right injection* above confirmed	Resectable	Left pneumonect	Pulmonary involvement sufficiently distal to allow resection
16	731983	1 Pressure on SVC minimal	Resectable	Not resectable	Extensive node metastases about upper hilus and in lt paratracheal area, SVC pushed forward
17	611886	1 Lt PA occluded close to origin 2 Defect lt atrium 3 PA separated from aorta	Not resectable	Not resectable	Massive nodal involvement of hilus and superior mediastinum
18	152762	Poor visualization of superior branch, rt pulmonary artery and PV	Resectable	Right pneumonect	PA ligated within pericardium, sup PV occluded to 1 cm from left atrium
19	911234	1 Narrowing of SVC 2 Occlusion first branch rt PA	Not resectable	Not resectable	Tumor from RUL to mediastinum with pressure on SVC, extensive nodes about PA
20	412021	Right sup mediastinal fullness produced by tortuous rt innominate vein and artery	Resectable	Left upper lobectomy	Small peripheral tumor in left upper lobe
21	357396	1 Displacement and ? invasion of SVC 2 Displacement of branches of RPA	Not resectable	Not resectable	Large tumor involving RUL and mediastinum with fixation
22	916423	1 Constriction of left PA at origin	Not resectable	Left pneumonect	Extensive tumor with narrowest of margin of division of PA, lt atrium invaded but segmentally removed
23	954223	Peripheral tumor not involving vessels	Resectable	Left upper lobectomy	Primary lesion LUL, soft involved lymph nodes between PA and bronchus prevented pneumonectomy

TABLE I. ANGIOCARDIOGRAPHY IN DIAGNOSIS OF RESECTABILITY OF CARCINOMA OF LUNG

	MGH UNIT	INVOLVEMENT BY ANGIOCARDIOGRAPHY	PREDICTION	RESULT	OPERATIVE FINDINGS
1.	891969	1. Obstruction left pulmonary artery 2. Defect left atrium	Not resectable	Not resectable	Tumor encircling pulmonary artery and lying anterior to hilus
2.	911545	1. Superior vena cava distorted 2. Occlusion right PA, superior branch	Not resectable	Not resectable	Carcinoma involving vena cava, pulmonary artery and vein
3.	860017	1. Invasion of SVC 2. Right pulmonary artery obstructed	Not resectable	Not resectable	Extensive lymph node metastases occluding SVC
4.	661031	Upper lobe branches, left PA distorted and small	Resectable	Left pneumonect.	Tumor close to pulmonary vessels and aorta
5.	799470	Aortic arch separated from LPA by mass	Resectable	Left pneumonect.	Large tumor about LPA, adherent to aorta and pericardium; left innominate vein invaded
6.	819044	No involvement of essential vascular structures	Resectable	Right pneumonect.	Hilus free; paratracheal nodes enlarged but not by tumor
7.	923419	1. Compression of LPA near origin 2. Tumor pressing on aortic arch 3. Defect left atrium	Not resectable	Not resectable	Extensive tumor about hilus; positive prevascular nodes
8.	817615	1. Sparse filling of LPA but no occlusion	Resectable	Left pneumonect.	1/2 cm. between origin of LPA tumor adherent to outside of PA
9.	933298	Displacement and poor filling of RPA without occlusion	Resectable	Right pneumonect.	Involved lymph nodes behind UL artery; RPA ligated within pericardium
10.	811557	1. Distortion rt. innominate vein and SVC 2. First branch RPA occluded 3. Defect left atrium	Not resectable	Not resectable	Massive involvement of hilus, heart and all great vessels
11.	864448	1. Superior vena cava displaced and irregular 2. Defect left atrium	Not resectable	Not resectable	Extensive metastases about upper hilus and trachea
12.	749995	1. Pressure on inferior branches of left PA 2. Left inferior PV and left atrium involved	Not resectable	Left pneumonect.	Inferior PV invaded by tumor but removed

even at a point quite close to the main pulmonary trunk, carries a much less ominous interpretation, because the tumor or a large lymph node may merely be resting against the artery and not actually invading it

2. Occlusion or severe narrowing of the branches of the right pulmonary artery to the upper or lower lobes within 1 cm. distal to the bifurcation of the right pulmonary artery (Fig. 1). This point lies slightly more distal than considered the point of inoperability by Dotter and Steinberg It lies just outside the pericardium. Occasionally it has been possible to ligate the right pulmonary artery more proximally by opening the pericardium.

Fig. 2.—Lateral view of superior vena cava and pulmonary artery, showing anterior displacement of the vena cava produced by lymph nodes invaded by tumor. Note nodularity.

3. Partial or complete obstruction of the innominate veins or the superior vena cava. It is quite possible to resect such structures, replacing them with venous grafts, but to date this has not been found advisable.

In addition to the overt degrees of involvement described above, very minor deformities indicative of pressure have proved significant. A tumor which presses on the superior vena cava or innominate vein may invade its wall and preclude resection, in contrast with a deformity of similar magnitude on an artery. This is particularly true if the indentation of the vein be nodular in character (Fig. 2), or if the vein be separated from normally adjacent structures (Fig. 3). It is recognized that interpreting pressure c in each instance normal temperature was present by the following morning. In another 2, urticaria appeared within an hour; in each the hives rapidly disappeared following the administration of oral Pyribenzamine. One patient noted generalized malaise for approximately 48 hours. There were no known late sequelae. In particular there was no indication of significant thrombophlebitis.

During the examination, patients experience a sense of warmth from head to toes. They become flushed and occasionally restless. There is a strong urge to cough. Within a few minutes they feel quite normal.

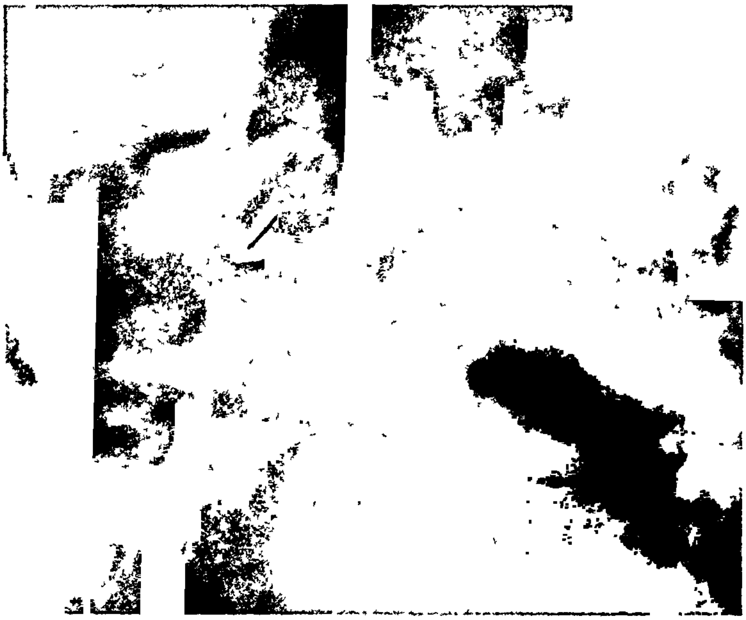

Fig. 1.—Tapered narrowing of the superior branch of the right pulmonary artery distal to its origin (arrow).

RESULTS OF STUDY

The following angiocardiographic evidence has been found of value in predicting unresectability.

1. Occlusion or a severe degree of narrowing of the left pulmonary artery within 1.5 cm. of its origin from the undivided pulmonary artery. Such involvement precludes separation of the branch vessel from the trunk. This corresponds with the experience of Dotter and Steinberg. Complete occlusion of the vessel usually is shown by a tapering or pointed ending seen best in the lateral projection. Certainly the length of the artery proximal to its point of involvement can be measured best in this projection or in the left anterior oblique position. Complete obstruction may be the result of other conditions and it is recognized that the finding is not direct evidence but, coexistent with pulmonary cancer, such findings are strong circumstantial evidence of malignant invasion. Narrowing of the artery in a concentric fashion also suggests, although less strongly, unresectability. Pressure upon one side of the artery,

caused by cancer is an assumption, but one which is usually justifiable in the presence of known primary carcinoma of the lung and in the absence of associated pulmonary sepsis, which might produce inflammatory enlargement of nodes

4 Involvement of the major pulmonary veins or the left atrium Normally the left atrium is smooth and regular in outline but in 6 cases observed in this series an irregular filling defect, usually measuring 1 to 3 cm in diameter, was visible in the heart chamber In every instance this occurred on the same side as the primary tumor, most commonly at the superior angle near the point of entry of the superior vein (Fig 4), but in one case at the point of entry of the inferior pulmonary vein At times the vein adjacent to the involved atrium was also invaded Care must be exercised in evaluating impairment or absence of visualization of a major pulmonary vein alone when the corresponding pulmonary artery is seriously embarrassed by tumor If the artery is well outlined however, and the vein is poorly filled with contrast material, involvement of the vein is strongly suggested, especially if there be an adjacent filling defect in the atrium

Pericardial invasion by tumor has been recorded by Dotter and Steinberg but was not seen in our series except as a finding coincident with atrial invasion

In our experience, preoperative prediction of impossibility of resection has been quite accurate Thirteen cases were deemed unresectable by angiocardiography Eleven of these were considered unresectable at thoracotomy In two instances a pneumonectomy was performed These patients died of residual carcinoma 6 and 7 months following pneumonectomy

In Case 12, the left inferior pulmonary vein was seen peripherally but not proximally and there was the suggestion of invasion of the left atrium at the entrance of this vein The removed lung showed tumor invasion of the inferior pulmonary vein

In Case 22 the angiocardiogram showed involvement of the left pulmonary artery very close to the main pulmonary trunk There was pressure superiorly on this common trunk Failure to demonstrate the left superior pulmonary vein was attributed to arterial obstruction The lung could be resected by division of the left pulmonary artery flush with the common trunk but tumor extended within 1 cm of this margin in the resected specimen Tumor grew along the pulmonary veins and invaded the left atrium so that a small segment of atrium had to be resected with the tumor

It was not the primary aim of this study to predict operability Indeed this phase of the problem was less successfully met, but errors in this direction are considered less serious, since for the patient's sake it is much better to err in this direction than in the opposite one Ten cases were predicted as operable by angiocardiography Seven were confirmed at operation, 3 proved to be inoperable

Case 14 had a tumor lying in the posterior thoracic gutter near the descending aorta Angiocardiography proved the mass to be separate from the aorta but failed to reveal involvement of the aorta by the tumor Exploratory thoracotomy disclosed this invasion of the aorta as well as the chest wall by the tumor

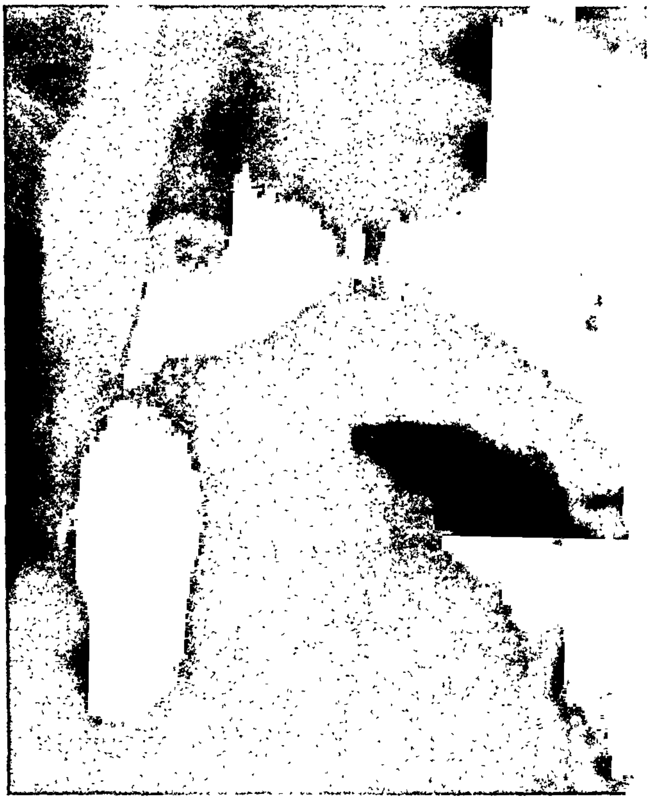

Fig. 3.—Vena cava, azygos vein, and trachea separated by enlarged azygos lymph node.

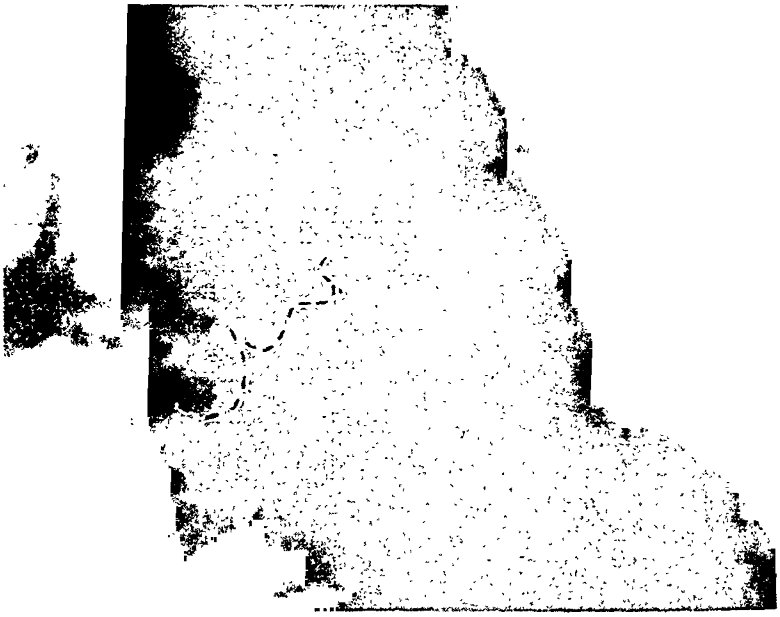

Fig. 4.—Tumor invading the right superior angle of the left atrium (dotted lines). Right superior pulmonary vein is not opacified.

A SIMPLIFIED METHOD FOR ACHIEVING INTRARESPIRATORY HUMIDIFICATION IN THE TRACHEOTOMIZED PATIENT

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IN THE CARE of severe respiratory conditions tracheostomy is often resorted to for varying lengths of time in order to maintain an unobstructed and patent airway in the acutely ill patient. However, the problem of intrabronchial crusts arises frequently as a complication of tracheostomy. This problem has been especially common in those patients with severe neurological diseases such as poliomyelitis, head injury, and in crush injuries of the chest.

The basic problem in the prevention of intrabronchial crusts is the prevention of inspiration of secretions by maintaining adequate tracheobronchial humidity.[1] Various methods of achieving this have been tried. One has been the direct instillation of irrigating fluids into the tracheotomy tube. These fluids are then carefully aspirated in the paralytic patient by means of semisterile technique with the realization that deep suctioning might easily traumatize the respiratory tree and cause additional mucous plug formation. The nonparalytic patient is encouraged to cough up the instilled fluids which are then suctioned out of the tracheotomy tube. The most frequently used liquids have been detergents such as Duponol C,* and Alevaire,† or isotonic saline. The use of these irrigations has resulted in a distinct decrease in mucous plug formation. The irrigations often float out the small mucous plugs before they become sufficiently enlarged to cause partial or complete obstruction of the lesser bronchial radicals. Other investigators have used herb teas, mucolytic enzymes in a suitable vehicle and drugs administered systemically in order to thin secretions.

Another method is to administer water vapor or water droplets. The warm steamer has provided more problems than solutions since constant supervision is necessary to prevent burns to the trunk, neck and respiratory tree. Moreover, proper humidification of the tracheotomy is difficult. The cold steamer and nebulizer have proved to be more efficacious, but require fairly elaborate equipment for attachment to the tracheotomy tube. The conventional bottle bubbler attached to most oxygen administration units provides little increase in tracheobronchial humidity. Preheating the water and

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*Duponol = C (sodium lauryl sulfate).

†Alevaire, Winthrop Laboratories.

Case 16 had a mass in the right upper lobe extending from the hilus to the chest wall. Angiocardiography showed minimal pressure on the posterior aspect of the superior vena cava below the azygos vein, but this pressure defect appeared to change and was not deemed sufficiently conclusive to warrant a diagnosis of tumor involvement. The right pulmonary artery filled slowly and seemed small but was without evidence of localized involvement. Thoracotomy disclosed the tumor adjacent to but not invading the chest wall. Lymph nodes in the mediastinum and at the right hilus were involved by tumor and pushed the superior vena cava forward without obvious invasion of this vessel.

Case 23 had a peripheral tumor in the left lung without evidence of metastasis to hilar or mediastinal nodes by conventional roentgenography or angiocardiography. Thoracotomy disclosed many grossly positive but very soft lymph nodes lying in front of the aorta, behind the pulmonary artery and between the latter and the left main bronchus. The adherence of these nodes to vessels prevented pneumonectomy.

The number of cases is obviously too small to be of any statistical importance, but the types of vascular distortion are nevertheless interesting. We have been impressed particularly by the grave significance of even minor degrees of distortion of the superior vena cava. Equally striking has been the frequency of involvement of the left atrium, which occurred nearly as often as superior vena cava deformity. These changes, which have not been stressed previously, can be appreciated only if multiple films, of good quality, are obtained, thus permitting the observer to be certain of the constancy of findings, which may be overlooked or disregarded on a single film.

SUMMARY

1. Experience with preoperative prediction of resectability of 23 cases of primary carcinoma of the lung studied by angiocardiography at the Massachusetts General Hospital is presented.

2. Thirteen cases were considered unresectable, and this was confirmed in eleven instances.

3. In 10 cases, no evidence was found which indicated involvement of heart and great vessels, and 7 were so found at operation.

4. Unnecessary thoracotomies may be avoided in cases demonstrated as unresectable by this method.

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local requirements of the lung A similar drip directly into the tracheotomy tube has been found to have the same beneficial effect of thinning the secretions even when the oxygen was not concurrently given The isotonic saline bottle is marked "For Tracheotomy Tube Use Only" as is the regulating clamp in order to avoid confusion with the intravenous solution bottles in order to prevent accidental drowning of the patient * Second, the bottle is kept at the level of the patient's head in order to reduce hydrostatic pressure and to prevent further the possible overly rapid administration of the isotonic saline solution

Results over the past 4 years have shown that intrabronchial crusts definitely have been diminished The conscious patient with a cough reflex can raise sputum with much less difficulty, thereby decreasing the endotracheal suction traumatization and ensuing complications of intrabronchial plugs, infections, and atelectasis

SUMMARY

A simple technique is described for the maintenance of adequate tracheobronchial humidity in tracheotomized patients This consists of a slow drip of isotonic saline through a 25 gauge needle into the lumen of an oxygen catheter Adequate humidification is then obtained with the prevention of intrabronchial crust formation The required equipment is readily obtainable

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*Such an accident would be unlikely in view of the fact that in the normal individual up to 1 L of physiological saline can be instilled into the lungs in a 24 hour period without ill effects bubbling the oxygen through additional lengths of multiholed tubing become useless unless the unit is attached within a foot of the tracheotomy tube because the change in temperature decreases the relative humidity.

Lung air contains about 6 per cent water vapor; inspired air contains about 1 per cent water vapor and oxygen, less than 1 per cent water vapor. To meet the above standard of water vapor content, a simple device was desired that could be attached to all tracheotomized patients, whether in a respirator or surrounded by other cumbersome equipment, without fear of drowning the patient and still providing adequate humidification.

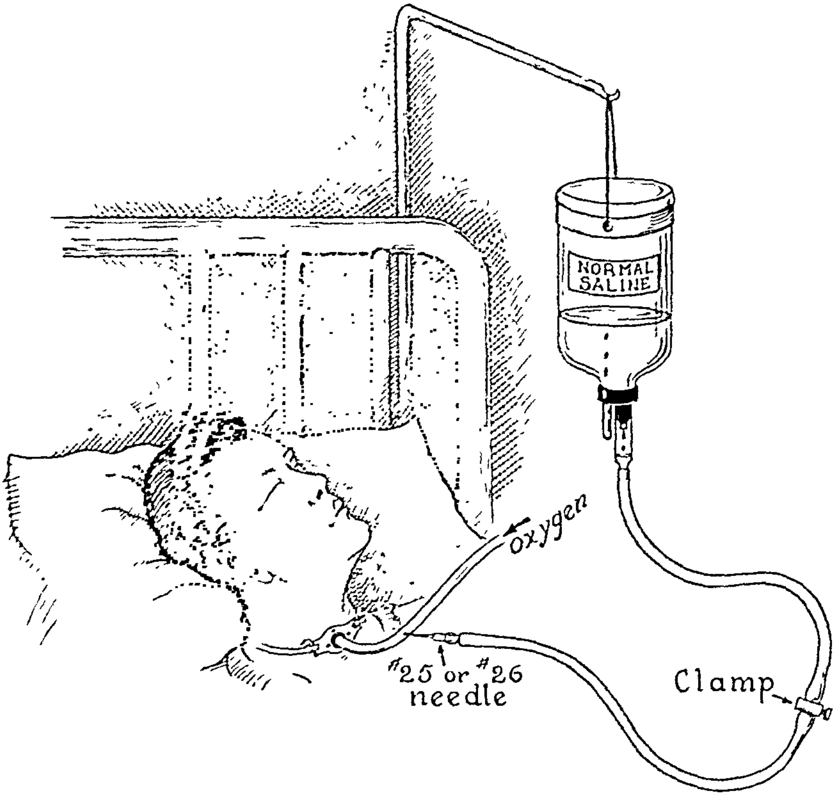

Fig. 1.—A drawing which depicts the relative simplicity of the apparatus required to achieve an adequate degree of intrabronchial humidification in the tracheotomized patient.

In experiments conducted by Basch and co-workers[3] regarding the influence of drugs and agents on tracheobronchial secretions, water vapor liquefied the secretions most efficiently. We, therefore, selected isotonic saline as the most readily available sterile solution which would satisfy the requirements of humidification.

The isotonic saline is administered by the usual intravenous tubing with a No. 25-gauge needle which is placed in the lumen of the oxygen-administration tube about 6 inches from the tracheotomy tube. The isotonic saline flow is regulated with a Hoffman clamp at 4 drops per minute. At an oxygen flow rate of 7 L. per minute the oxygen stream vaporizes the saline and disperses the larger droplets into the trachea. These droplets migrate into the respiratory tree and vaporize according to the temperature of the patient and to the

Bronchoscopy on the fifth and twelfth hospital days revealed granulation tissue obstructing the distal portion of the bronchus intermedius. Bacterial culture showed aerobic Klebsiella which was strongly sensitive to Chloromycetin. No growth was obtained from cultures for acid-fast organisms and fungi. Biopsies of the area showed chronic inflammation of the wall of the bronchus. The surface was covered by a layer of necrotic tissue and fibrinopurulent exudate in which one could see many broad, irregular, nonseptate hyphae (Fig. 2). The fungus was almost entirely in necrotic tissue. The hyphae were basophilic and readily visible in routinely stained tissue. Bacterial stains showed numerous gram-positive cocci but no acid-fast organisms were seen.

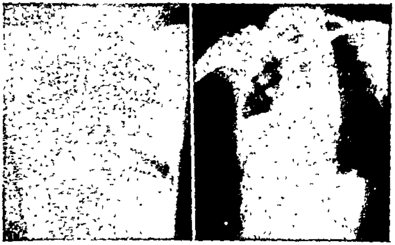

Fig. 1.—A chest film taken the day of admission shows consolidation of the right lower lung field. The air-fluid level is seen in the oblique view taken the day after admission. This persisted without change for [illegible] days.

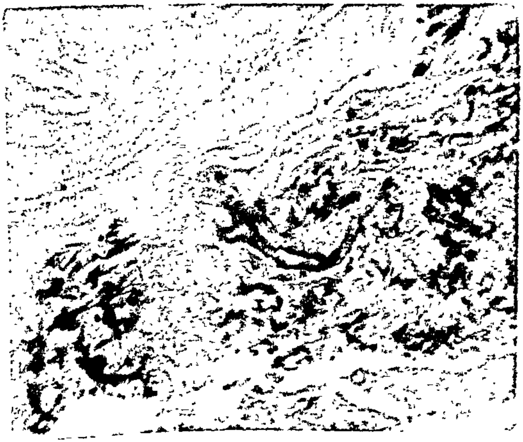

Fig. 2.—Bronchoscopy biopsy specimen. The section shows a branching, nonseptate hypha in necrotic material. (Hematoxylin and eosin; ×221; reduced [illegible])

Following the second bronchoscopy, she became worse with daily elevations in temperature ranging from 38° to 39° C. She continued about the same to the twenty-fifth hospital day, despite antibiotics, and frequently needed supplemental doses of regular insulin. Serial chest films remained essentially unchanged with the air-fluid level remaining in the right lower lung field.

MUCORMYCOSIS OF THE BRONCHUS SUCCESSFULLY TREATED BY LOBECTOMY

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Until recently, mucormycosis infection was considered a uniformly fatal disease. Harris,[1] from this clinic, reported a diabetic patient with nasopharyngeal mucormycosis in whom the diagnosis was made during life. Recovery followed rigid control of diabetes, desensitization against the fungus, and administration of iodides.

The authors have treated a second patient in whom the diagnosis of mucormycosis was made during life. The factors which led to a satisfactory recovery of this patient form the basis for this report.

CASE REPORT

R. G. (Duke No. E30177) was a 20-year-old Negro woman who had had diabetes for at least 5 years. Two and one-half months prior to her admission to Duke Hospital she was treated at her local hospital for her first episode of diabetic coma and acidosis. A second episode, 6 weeks prior to her admission to Duke, responded well to the usual therapy for acidosis. On the sixth day of this admission she developed an elevation of temperature to 39.5° C. An area of consolidation in the right lower lung field was demonstrated by x-ray examination. In spite of penicillin therapy she did not improve and was admitted here on March 30, 1956.

On admission the temperature was 37° C., pulse 100, blood pressure 108/60 mm. Hg, and respiration 22 per minute. The trachea was shifted to the left. Over the right lower chest the breath sounds, tactile fremitus, and vocal fremitus were decreased, and there was dullness to percussion. The liver edge extended approximately 2 fingerbreadths below the costal margin and the organ was tender. The remainder of the physical examination was normal.

Laboratory studies on admission showed the hemoglobin to be 11.6 Gm., the white cell count 9,050 with 53 per cent polymorphonuclear leukocytes, 3 per cent stab forms, 2 per cent eosinophils, 10 per cent basophils, 3 per cent small lymphocytes, 22 per cent large lymphocytes, and 16 per cent monocytes. Urine examination showed a specific gravity of 1.034 and a pH of 5. There was no protein or acetone, but 4+ sugar was present. Microscopic examination of the urine was negative. *Candida albicans*, nonhemolytic *Micrococcus pyogenes* var. *aureus* and *Neisseria sicca* were grown from the sputum. Gram stain of the sputum showed a predominance of gram-negative diplococci.

A fasting blood sugar was 222 mg. per cent and serum cholesterol 266 mg. per cent.

Skin tests for old tuberculin 1:1000 was 2+ in 48 hours. Skin tests for blastomycin, coccidioidin, and histoplasmin were negative.

A chest film showed complete density of the right lower chest (Fig. 1). During the first week, her diabetes was well controlled and she began coughing up foul-smelling, purulent sputum. Fluoroscopy revealed an air-fluid level in the right lower lung field.

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Sections through the bronchus intermedius and adjacent lymph nodes showed marked necrosis of the bronchus with infiltration of the bronchus by nonseptate hyphae (Figs. 4 and 5).

She was discharged on the forty third postoperative day. The diabetes was controlled on a 2,100 calorie diet with 52 units of NPH insulin with 5 units of regular insulin administered in the morning. A vaccine made up of *Candida albicans* and Mucor, 1:1000 dilution, had been started while in the hospital to be continued three times a week by her county health office.

Although she has not returned here for follow up, a report from her county health office states she continued to do well for 6 months following discharge and then moved out of the state.

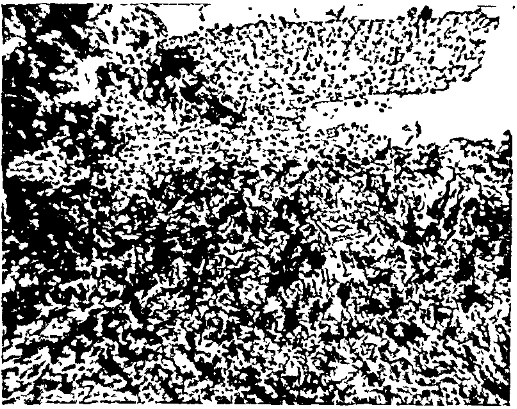

Fig. 5.—Section from bronchus of operative specimen. The section shows a matted mass of hyphae lying on the epithelial surface of the bronchial wall. (Hematoxylin and eosin; ×167, reduced ¼.)

DISCUSSION

Pulmonary mucormycosis has been reviewed recently by Baker.[2, 3] In his series of patients, all of whom died, the disease occurred only in those who had concomitant disease that could have reduced the resistance of the body to infection. Diabetes mellitus, multiple myeloma, leukemia, infant diarrhea, and thermal burns were among the predisposing factors.

This is the first reported patient who has had a pulmonary resection for mucormycosis and the second patient in whom a diagnosis has been made during life. From similar patients, previously reported, it is apparent that a pre-existing severe illness is necessary for the establishment of pulmonary mucormycosis. In our patient, diabetes mellitus with acidosis and coma was the predisposing factor in reducing resistance to infection. With the marked inflammatory reaction and destruction of the bronchial wall, obstruction was produced. This then favored the development of a bacterial abscess distally.

The principal factor necessary to effect a cure of a patient with mucormycosis is to control the pre-existing disease as advocated by Harris.[1] Removal of the nonareated destroyed lung in our patient was a second essential factor, On the twenty-fifth hospital day, resection was attempted but postponed because of aspiration of secretions during intubation. This cleared and on the thirtieth hospital day she was intubated under topical anesthesia with a Carlen's bronchospirometer tube and, under general anesthesia, a right middle and lower lobectomy was performed. A tracheostomy was done at the end of the procedure to maintain tracheobronchial toilet. She did well and became afebrile on the seventh postoperative day. Antibiotics were continued for 10 days after operation. Serial films following the middle and lower lobectomy on the right showed the left lung and right upper lobe to be expanded well (Fig. 3).

Fig. 3.—A chest film made 5 weeks after right middle and lower lobectomy shows good expansion of the remaining upper lobe with some postoperative pleural reaction at the right base.

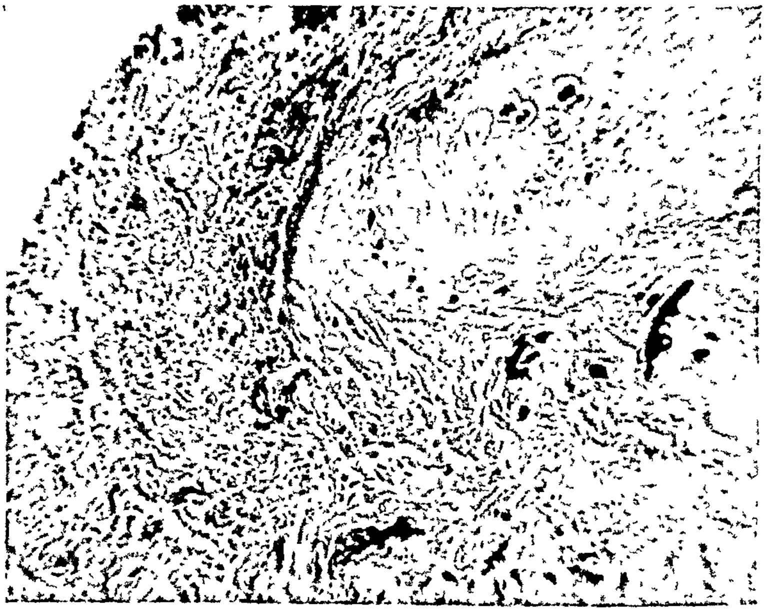

Fig. 4.—Section from bronchus of operative specimen. The section from the wall of the bronchus intermedius shows hyphae extending deep to the bronchial cartilage. (Hematoxylin and eosin; ×167; reduced ⅕.)

Gross examination of the lobes removed showed a large abscess containing 30 to 40 c.c. of sanguino-purulent material in the lower lobe, with atelectasis and consolidation of the middle lobe. Sections taken from the superior segment of the lower lobe, basilar portion of the lower lobe, through the parenchyma of the middle lobe, and from the wall of the abscess of the lower lobe showed only chronic nonspecific pneumonia with abscess formation.

ACCESSORY LUNG WITH PERSISTENT LEFT SUPERIOR VENA CAVA AND DUPLICATION OF INTESTINE

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In comparison with other malformations of the lung, accessory lung is an uncommon observation. Herxheimer,[1] in 1901, described a normally aerated accessory lung. He found a small functional "third lung" stemming from a tracheal bronchus 2.5 cm. down from the cricoid cartilage. This accessory lung "set above the lung on the right side" had caused stenosis of the trachea when it pressed against the right side of this structure. However, he did not describe the origin of the blood supply to this accessory lung. The second case of true accessory lung was reported by Muller.[2] This, like that of Herxheimer, was a right sided accessory lung originating from the trachea. Again details of blood supply are absent even though he describes an artery and a vein to the accessory lung on serial microscopic sections. Tracheal diverticula and tracheal accessory lungs were discussed many years ago by Chiari.[6] In 1940, Hulse and Curtis[3] reported a case of a true tracheal accessory lung which was diagnosed by bronchogram. Cotton, Spaulding, and Penido[4] reported a whole true accessory lung with three lobes located in the right thorax. This was said to be the first instance of a true accessory lung removed by surgery. The blood supply was a single branch of the subclavian artery. The upper, accessory cystic lung came directly off the trachea by a stem bronchus that measured approximately 3 mm. in diameter and 1 cm. in length. In 1953, Seley[5] reported what he said to be the first recorded instance of an accessory lung situated in the suprahilar area receiving an artery from the aorta and showing evidence of cystic disease and sequestration.

The purpose of this report is to define and discuss a case of pure or true accessory lung. This malformation is to be clearly delineated from other conditions with which it is confused. Loosely defined terms such as ectopic, supernumerary, aberrant, lower accessory lobes, tracheal diverticula, bronchogenic cysts, etc., have confused the issue of the exact morphology of true accessory lung. The case presented in this report afforded an opportunity to inject the arterial and venous blood supply to the accessory lung and thereby trace the exact origin and course of these vessels.

CASE REPORT

A one month old white female child was admitted to the Medical College of Virginia Hospital on June 5, 1956, because of chronic constipation, straining at stool and the recent

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for this removed an area that harbored an infection and made control of the diabetes possible. Invasion of blood vessels of the lung by the Mucor with resultant infarction, as so frequently occurs, was not found in our patient. The limitation of fungus infection to the bronchus, followed by normal healing of the bronchial stump was, we felt, due to the rigid control of her diabetes.

The fungus was not grown from this patient possibly because the number of fungi were few as compared to the number of other organisms. The fungus in the tissue was confined to a small segment of the bronchus and was not seen in the cavity in the lung. The cavity distal to the site of fungus infection was a typical pyogenic abscess such as one usually sees with bronchial obstruction. The fungus could not be identified in the wall nor could one see the usual reaction associated with the presence of this fungus, namely, necrosis of tissue with little or no cellular infiltrate. The organism would surely have grown if a portion of the bronchus had been used as inoculum. Phycomycetes are readily recognized in tissue sections by the masses of nonseptate hyphae and, when invasive into the tissue, may be considered pathogenic.

SUMMARY

This is the first case of pulmonary mucormycosis in which a pulmonary resection was performed, and the second case in which the diagnosis was made during life.

The pathogenesis and course is discussed.

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Following admission, the infant was placed on a Similac formula. She took feedings fairly well for the most part with variable amount of regurgitation. The weight had increased to 6 pounds, 4 ounces by the sixteenth hospital day when a laparotomy was performed.

Operative Findings:—On inspection of the peritoneal cavity, it was apparent that the terminal ileum was duplicated as well as the cecum and the appendix. There was a duplicated large intestine down to and below the peritoneal reflection (Fig. 1). The duplicated colon had a mesentery of its own from the terminal ileum up to the mid-transverse colon. At this point the wall became common with the normal colon. The duplicated colon was three times normal size, cystic in nature and its walls were definitely thickened. The duplicated terminal ileum was resected and the proximal line closed with silk sutures. The duplicated cecum, appendix ascending colon and proximal transverse colon which had its own mesentery was resected without difficulty and the distal orifice of the duplicated colon was exteriorized as a fistula.

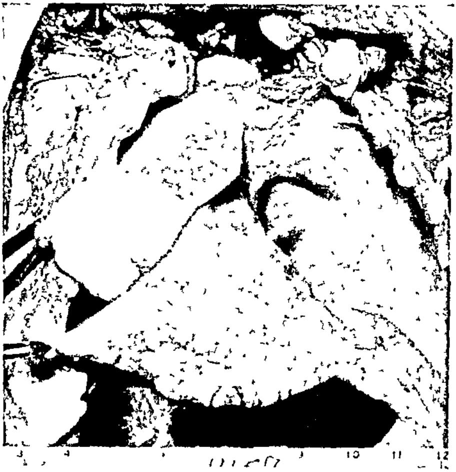

Fig. 2.—The two lobes of the left lung are reflected forward, revealing an aerated accessory lung lying in the posterior part of the thoracic cage and separated from the left lung by a translucent sheet of pleura

Postoperatively, the infant did satisfactorily until the eighth postoperative day when she developed vomiting and subnormal temperature. The respiration became difficult and there was marked inspiratory retraction with greenish mucoid material exuding from the nose. There was intense cyanosis and coarse rhonchi and râles throughout the chest. The patient died.

Autopsy Findings.—There was an accessory lung lying in the posterior portion of the left upper part of the pleural sac (Fig. 2). This lung possessed its own pleural cavity and like the other lobes of the lungs was well aerated. This latter finding accounted for the fact that no mass or lesion was seen by x ray. The accessory lung had a communication with the trachea by an eparterial bronchus which arose from the left side of the trachea less than 1 cm. above its bifurcation. The lungs with their six lobes and their contained blood supply were injected with vinylite plastic (Fig. 3). The result of this showed that the accessory lung was supplied on the arterial side by a branch of the left main pulmonary artery. The venous return was by the pulmonary veins. An additional anomaly was drainage of the azygos system into a left persistent superior vena cava (duct of Cuvier) which drained into the left atrium.

appearance of a mass at the anus. The infant was born one month prematurely and weighed 5 pounds and 6 ounces at birth. From the beginning, the infant ate an evaporated milk formula poorly and spit up a good deal of each feeding. The stools had always been small, hard, and yellow. Approximately one week prior to admission the infant began to strain more during defecation and at this time the mother noted a mass at the anal orifice. On the day before admission, she noted some bright red blood on the diaper. There had been no vomiting, fever, or other symptoms and the history was otherwise negative.

Physical Examination:—Temperature 98.6° F., weight 5 pounds, 13½ ounces, pulse 150, respiration 30. The general appearance and the physical examination were not abnormal except for the findings confined to the anus and rectum. On straining there appeared to be protrusion of the rectal mucosa on the left side of the anus and laxity of the perineum. Digital examination revealed a tubular cystic mass encroaching on the left side of the anus, rectum and rectosigmoid. The mass was between the rectum and pelvis. There did not appear to be any connection between the mass and the sacral and retroperitoneal region posteriorly, nor did there appear to be any connection by a stalk between the mass and the rectal mucosa higher up. The examining finger could not be inserted between the mass and the external circumference of the anus. The lumen of the anus and rectum appeared adequate for bowel movement.

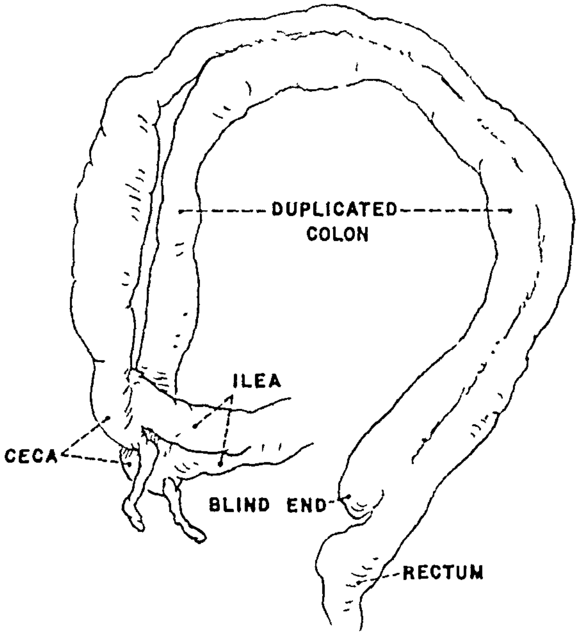

Fig. 1.—Duplicated small and large intestines.

Laboratory Data:—The urinalysis was negative. Hemoglobin 15.2 Gm.; white blood cells 17,800 with 42 per cent polymorphonuclears, 1 per cent eosinophils, 25 per cent lymphocytes and 32 per cent monocytes. Smear showed a strong shift to the band stage and severe toxic changes. Serum sodium 145, potassium 6.0 mEq./L., chlorides 97 mEq./L., and CO_2 22 mEq./L., respectively. BUN was 17 mg. per cent.

A barium enema on the day after admission revealed a very redundant but normal-sized colon with questionable malrotation of the cecum. Filling was not completely satisfactory. Evacuation was poor. Roentgenograms of the chest revealed a normal cardiac shadow with slight prominence of the bronchovascular markings throughout and linear densities in the right upper lung "compatible with linear atelectasis or residual changes of pneumonia." A gastrointestinal study with Lipiodol was also negative.

it showed not only duplication of the lungs, but also intestinal duplication The anomalous or persistent venous return to the left atrium and the left persistent vena cava are of passing interest in this case

SUMMARY

A case of true accessory lung, duplication of intestines, and anomalous venous return to the left side of the heart is reported It seems probable that the accessory lung should be so designated only if the following criteria are fulfilled origin from the trachea or main stem bronchus, a pulmonary blood supply and a separate pleural cavity

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Anatomic Diagnosis.—Duplication of the terminal 2 cm. of ileum, cecum, appendix, ascending colon, transverse colon, descending colon, sigmoid colon, and upper rectum.

Surgical resection of duplicated terminal ileum, appendix, cecum, ascending colon, and proximal transverse colon.

Localized fibrinous peritonitis of colostomy site.

Colostomy, anterior abdominal wall, involving proximal end of duplicated large bowel not surgically removed.

Duplication of left lung—accessory left lung with its own pleural cavity.

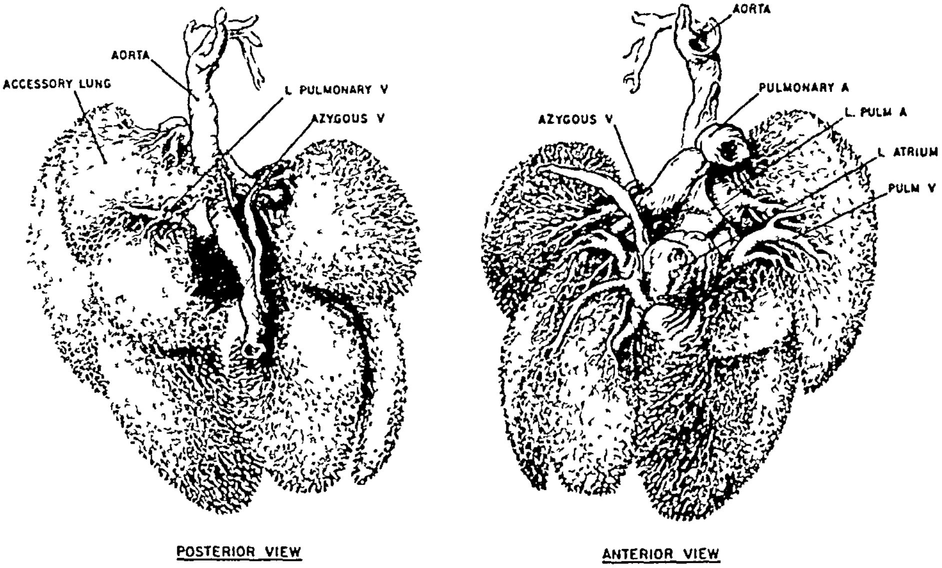

Fig. 3.—The vinylite plastic injection according to the method of Liebow. The accessory lung is supplied by a pulmonary artery and drained by a pulmonary vein. There is no connection with the aorta.

DISCUSSION

There appears to be little or no difficulty in defining the anatomic features of such pulmonary anomalies as azygos lobes, bronchogenic cysts, congenital cystic disease, mediastinal cysts, and tracheal diverticulum. Review of the literature quickly demonstrates the confusion between terms such as true accessory lung, pulmonary sequestration and pulmonary sequestration of accessory lobes and, finally, aberrant and heterotopic lung. In our opinion, true accessory lung is composed of two or more lobes, is contained in its own pleura, and derives a bronchus from either the trachea or the main-stem bronchus.

Furthermore, both the arterial and venous blood supplies are entirely pulmonary in derivation and course. In other words there is no supply from the systemic arteries. The aberrant lobes have no connection with the trachea or bronchi and the blood supply is from the systemic artery. Pulmonary sequestration is a malformation characterized by "cutting off" of the developing lung and, as would be expected, possesses a systemic blood supply. From a review of the literature and our own case we are of the opinion that the development of a true accessory lobe is *not* brought about by the mechanism of pulmonary sequestration. It is more accurately a duplication or supernumerary process. It is our feeling that the case presented in this report supports our view in that

Laboratory studies showed albuminuria and a blood urea nitrogen of 27 mg per cent, hemoglobin 13.1 Gm per cent, hematocrit 45 per cent, white cells, 6,650, with a normal differential, microscopic examination of the urine was negative.

A left thoracotomy was performed under Pentothal nitrous oxide ether oxygen endotracheal anesthesia. During anesthesia there was a fall in blood pressure which was controlled by a vasopressor given in continuous intravenous drip.

Palpation of the left lung at operation disclosed 2 distinctly separate masses in the left lower lobe. The first mass lay in the superior segment of the left lower lobe, was ovoid and firm, and was adjacent to the interlobar fissure. The second mass occupied the lateral basal segment of the left lower lobe. Large hard lymph nodes were present at the hilum

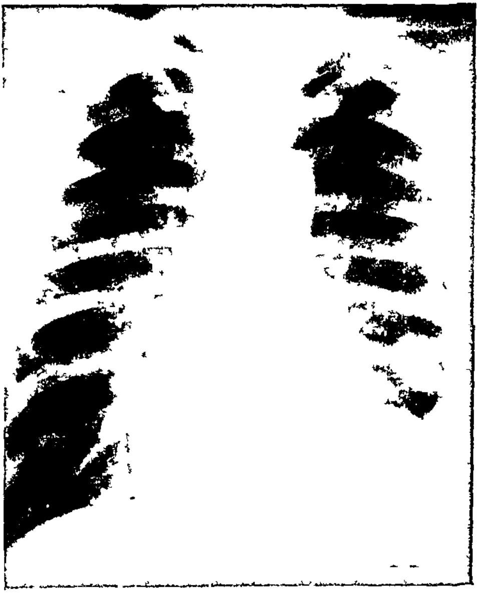

Fig. 1.—Roentgenogram of chest just prior to thoracotomy.

and in the inferior pulmonary ligament. Biopsy and frozen section of the mass in the superior segment was reported as epidermoid carcinoma. Because of the patient's cardi[illegible] status, pneumonectomy was deemed inadvisable. Left lower lobe lobectomy and hilar lymphadenectomy were performed. There was no cardiac or respiratory embarrassment postoperatively. The wound healed per primam and he was discharged on the ninth po[illegible]operative day.

Operative Specimen.—The specimen consisted of the lower lobe of a left lung an[illegible]ured 12 by 21 by 6 cm. The bronchus to the lateral basal segment contained in[illegible] mucus and could be traced into a circumscribed, nonencapsulated, ovoid, gray w[illegible] with focal areas of necrosis. It measured approximately 4.0 cm in diameter. T[illegible] to the superior segment of the left lower lobe was the site of origin of a[illegible]scribed, nonencapsulated gray white tumor measuring approximately 3.5 cm [illegible] were discrete and separated by an area of uninvolved parenchyma. Sev[illegible] nodes and a specimen labeled "node from lingula" were also received.

THREE PRIMARY CARCINOMAS OF THE LUNG ARISING IN A LEFT LOWER LOBE WITH METASTASIS OF TWO OF THE TUMORS

A Case Report

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INTRODUCTION

THE following case exhibited 2 primary tumors, composed of 3 histologically different variants of bronchogenic carcinoma, all arising in the lower lobe of the left lung. These variants were a well-differentiated squamous carcinoma forming keratin "pearls," a well-differentiated papillary adenocarcinoma, and an undifferenitated (oat cell) carcinoma. The latter 2 neoplasms produced metastases in regional lymph nodes and distant organs of "pure strains" or "lines" of tumor with no admixture or intermingling of the oat cell and adenocarcinoma in any of the metastases. There was no metastasis from the well-differentiated squamous carcinoma.

The case touches on continuing problems in clinicopathologic correlation and affords yet another example of unnecessary delay in a patient having clear indications for surgical intervention. When operation was permitted in this patient with significant cardiac disease, expert anesthesia and careful attention to detail permitted thoracotomy and lobectomy to be completed uneventfully.

CASE REPORT

A 62 year-old white man was admitted to The George Washington University Hospital for investigation of a lesion of the left lung noted on a roentgenogram. The patient had had a cough for 4 years attributed to chronic bronchitis. He had arteriosclerotic heart disease with several episodes of congestive failure controlled by digitalis, diuretics, and low sodium intake. He had recently been studied because of albuminuria, casts and hematuria, and a presumptive diagnosis of right renal vein thrombosis had been made.

Roentgen examination at another institution showed a shadow in the left lower lung field. He was advised by a physician to have nothing done about it because of his cardiac status. Six months later, the patient had some pain in the left chest area. Increase in the size of the lesion in the left lung could be seen in a second roentgenogram (Fig. 1), and surgical consultation was sought. Thoracotomy was advised despite the patient's heart disease since the pulmonary shadow was thought to be a tumor and there was no clinical evidence of distant metastasis.

Physical examination revealed a well-developed, well-nourished, white man. Blood pressure was 200/100 mm. Hg. Rhonchi were heard in the left chest below the scapula. The heart was enlarged to the left and auricular fibrillation was present.

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On microscopic examination, the tumor from the upper lesion (superior segment) was a well differentiated squamous carcinoma with "pearl" formation (Fig. 2, *A*). Its bronchial origin was seen microscopically and extensive areas of in situ carcinoma were noted adjacent to the infiltrating component. The lower mass (lateral basal segment) was composed of 2 distinct types of tumor: a peripheral portion, consisting of a papillary adenocarcinoma of bronchiolar origin (Fig. 2, *B*), and a proximal component showing an undifferentiated anaplastic carcinoma compounded of small ovoid to pyriform cells measuring 10 to 16 mμ with scanty cytoplasm (oat cell carcinoma) (Fig. 2, *C*). There was extensive vascular and lymphatic invasion by tumor with obliterative endarteritis and thrombosis (Fig. 2, *C*). There was a mingling of both elements at the mid portion of the lesion (Fig. 2, *B*), but relatively "pure" types proximally and peripherally. No zones of transition were found.

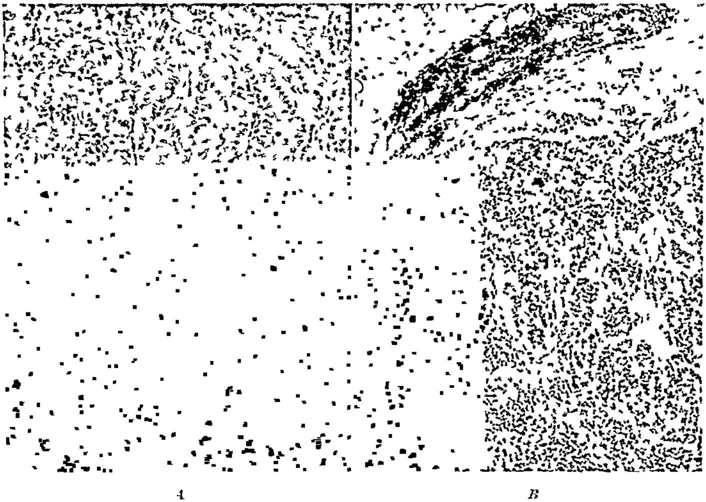
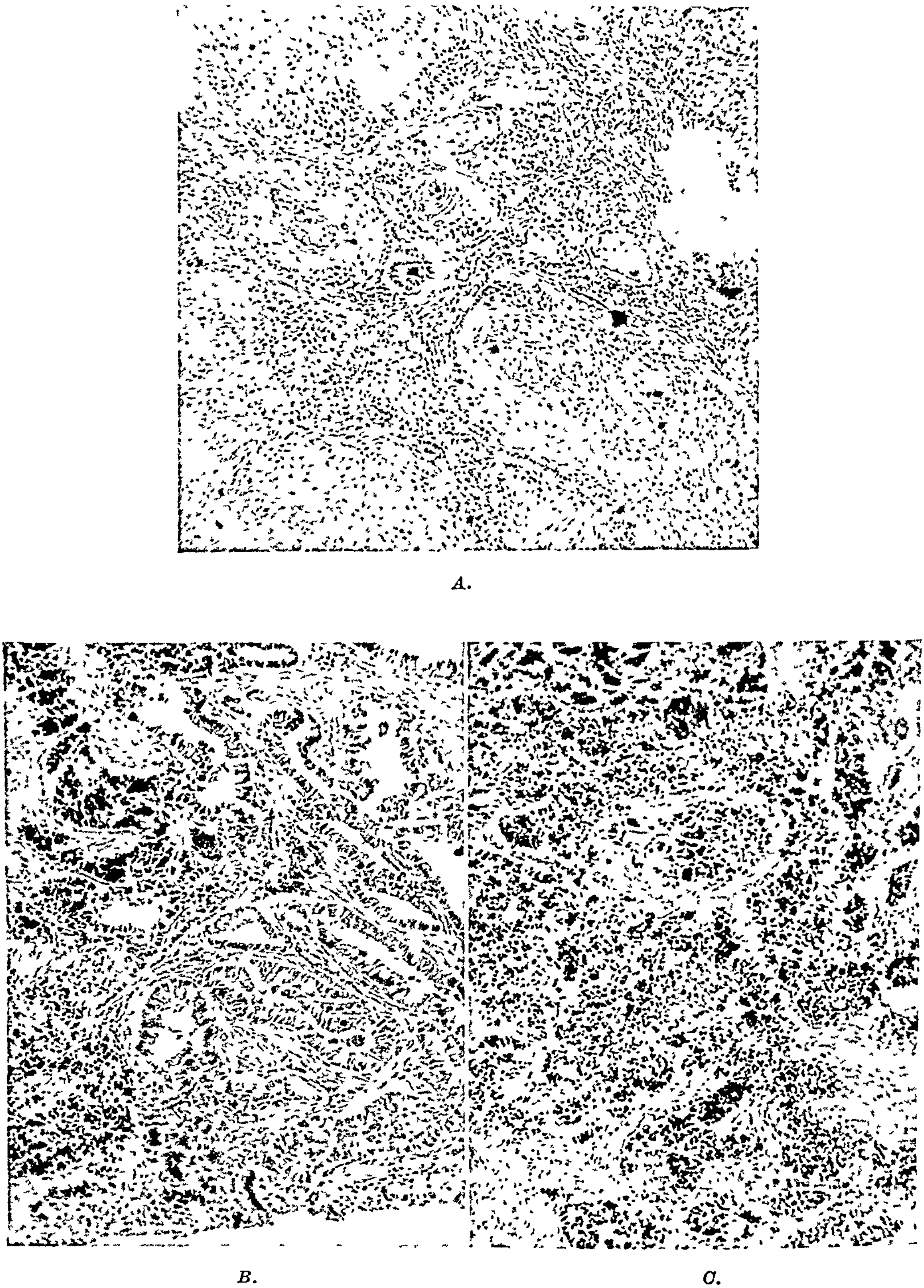


Fig. 3.—*A*, Lymph node, left hilum, containing metastatic adenocarcinoma. (Hematoxylin and eosin. ×120; reduced ½.)

B, Lymph node, left hilum, replaced in part by metastatic anaplastic carcinoma (oat cell). (Hematoxylin and eosin. ×120; reduced ½.)

Each of the elements had metastasized separately to hilar lymph nodes, some of the nodes containing metastatic papillary adenocarcinoma (Fig. 3, *A*), while others were in part or completely replaced by the "oat cell" element (Fig. 3, *B*). There was no intermingling or juxtaposition of cell types in any of the nodes. The well differentiated squamous carcinoma with keratin "pearls" from the superior segment was not found in any of the lymph nodes.

Subsequent Course.—Three months after operation, the patient slipped and injured his left hip. He was re-admitted to The George Washington University Hospital where x-ray examination showed a pathologic fracture of the neck of the left femur. Metastatic lesions were also found by x-ray studies in the ribs, left scapula, and right clavicle. The liver was noted to be enlarged and it was evident that the patient had lost considerable weight. Internal fixation of the neck of the left femur was performed under general anesthesia. Postoperatively, the patient became more feeble and was disoriented. He was discharged 3 weeks after this admission at the request of his family. He was admitted to another hospital one week after his discharge from The George Washington University Hospital and died on the following day.


A.

B. C.

Fig. 2.—*A*, Well-differentiated squamous carcinoma forming keratin "pearls" from superior segment of the left lower lobe. (Hematoxylin and eosin; ×100; reduced ⅕.)

B, Zone of admixture of well-differentiated adenocarcinoma and anaplastic (oat cell) carcinoma in lateral basal segment (Hematoxylin and eosin; ×120, reduced ⅕.)

C, Oat cell carcinoma, lateral basal segment, showing vascular invasion and obliterative endarteritis. (Hematoxylin and eosin; ×120; reduced ⅕.)

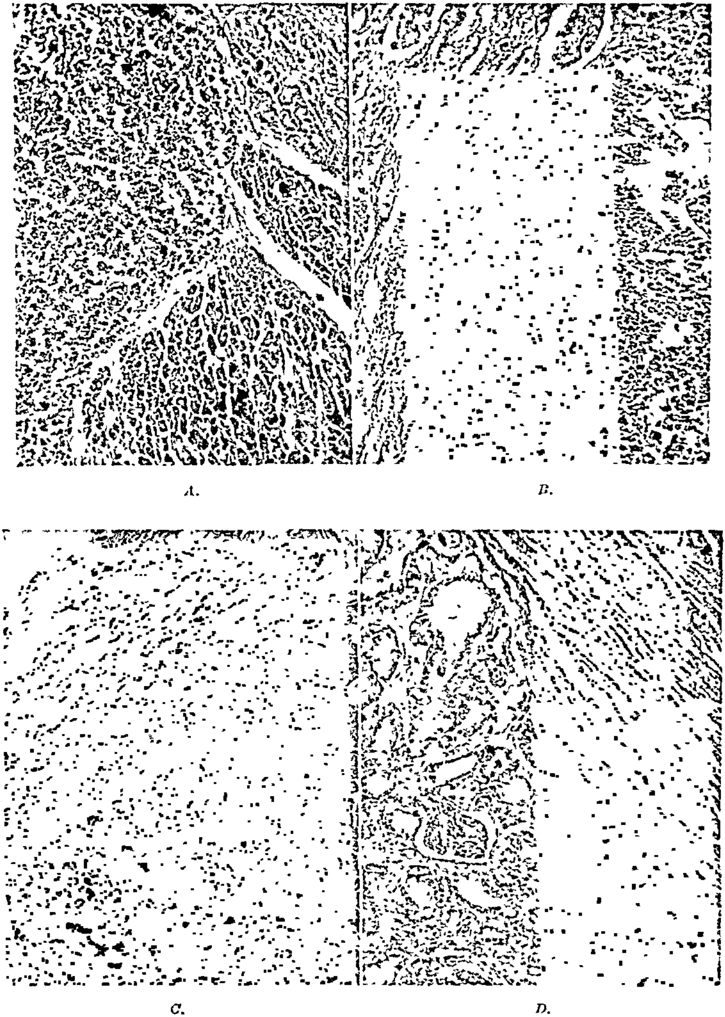

A. B. C. D.

Fig. 1.—A, Pancreas with metastatic oat cell carcinoma (Hematoxylin and eosin; ×90.)
B, Kidney with metastatic oat cell carcinoma. (Hematoxylin and eosin; ×90.)
C, Metastatic adenocarcinoma, right lower lobe lung. (Hematoxylin and eosin; ×30.)
D, Kidney with metastatic adenocarcinoma (Hematoxylin and eosin; ×90.) (All photomicrographs reduced ⅕.)

(We are indebted to Colonel J. E. Ash, M.D., pathologist, Suburban Hospital, Bethesda, Md., for a copy of his post-mortem findings and slides and permission to quote from his protocol.)

Summary of Pertinent Findings at Post-mortem Examination.—

No free fluid in thoracic or abdominal cavities.

Heart. 530 grams: Atheromatosis of aortic valve. Moderate degree of coronary sclerosis most marked in the circumflex branch of the left coronary artery. Area of scarring at apex of left ventricle suggesting old infarction. The interventricular septum contained a solitary metastatic nodule measuring 0.8 cm.

Lungs. Right 610 grams, left 530 grams: Right lung voluminous due to emphysema of the upper and middle lobes. Some collapse of the lower lobe with small nodules of metastatic tumor and a calcified tubercle. Hilar lymph nodes on the right enlarged and replaced by tumor. The remaining left upper lobe was firmly adherent to the parietal pleura and showed fibrous pleuritis. Many nodules of tumor were seen in the left hilar region.

Liver. 1,730 grams: Many umbilicated nodules of metastatic carcinoma, more pronounced in the right lobe.

Gall bladder: Metastatic tumor nodules in the wall of the fundus. Large tumor-bearing node related to the cystic duct, with no obstruction to biliary drainage.

Pancreas: The pancreas was the site of a tumor measuring 8 by 5 cm. which almost entirely replaced the body and tail of that organ and extended into the surrounding tissues with involvement of peripancreatic and coeliac nodes.

Kidneys. Right 180 grams, left 140 grams: Each contained multiple metastatic nodules in their cortices varying from 0.5 to 1.5 cm. The right renal vein was normal.

Gastrointestinal tract: Recent ulcer of the lesser curvature 1.5 cm. in diameter.

Great vessels: Moderate atherosclerosis of abdominal portion of the aorta. Metastases in para-aortic and retroperitoneal nodes.

Microscopic Examination.—

The separate "lines" of metastasis from the lower tumor found in the hilar lymph nodes at the time of the operation were still evident in autopsy sections of regional nodes and distant organs.

The bulk of the metastases were those of the "oat cell" component and were found in the pancreas (Fig. 4, *A*), kidneys (Fig. 4, *B*), periadrenal tissues, heart, gall bladder wall, and lymph nodes.

The papillary adenocarcinoma (bronchiolar component) was found in the right lower lobe (Fig. 4, *C*), right hilar lymph nodes and kidneys (Fig. 4, *D*).

There was no intermingling or admixture of the 2 components in lymph nodes or kidneys, each element, remaining separate in each of the metastases. Reticulum stains showed a moderate amount of stromal reticulin but none was produced by the tumor cells. Mucicarmine stains showed mucin in the (often papillary) adenocarcinomatous component, but none in the "oat cell" portion. There was no metastasis from the well-differentiated, keratin producing, squamous carcinoma of the superior segment of the left lower lobe (see Fig. 2, *A*) found in the lobectomy specimen. No section of liver was available for microscopic examination.

DISCUSSION

This case is unique in our experience by virtue of the "pure strains" or "lines" of tumor, with no zones of transition, found in the primary sites in the left lower lobe and maintained in regional node and distant organ metastases. It would appear to fit in the classification devised by Foulds as a rare instance of "independent primary tumors."[1] Some recent papers with photomicrographs away from zones of infiltration would seem to leave little doubt as to multicentric origin. This would appear to us to be a more useful basis on which to support the theory of multicentric origin of carcinoma of the lung than the variation in cell type. In the present case, extensive areas of in situ carcinoma were found in the bronchus of origin adjacent to the infiltrating portion of the well-differentiated squamous carcinoma in the superior segment of the left lower lobe.

Areas of in situ oat cell carcinoma and bronchiolar adenocarcinoma were also recognized away from areas of infiltration. The microscopic appearance of the tumors at these sites was reminiscent of some of the photomicrographs published in recent papers by Spain and Parsonnet[19] and Horrell and Howe[20] both of which lend strong support to the concept of the multicentric origins of carcinoma of the lung.

There seems to be a wider acceptance of this concept when applied to terminal bronchiolar adenocarcinoma than to the squamous and oat cell variants and, for this point, the large section mounts of McGrath, Gall and Kessler[2] offer strong additional evidence that all variants of bronchogenic carcinoma are multicentric in origin, as does the paper by Papanicolau and Koprowska.[21]

An impressive array of data supporting the concept of multicentric origin at many different sites in the body has been gathered and added to personally by Willis,[15] Stewart,[18] Slaughter[22, 23] and Collins and Gall.[24]

The clinical implications of the theory of the multicentric origin of cancer are manifold. Particularly pertinent to present-day modes of therapy are the margins in excisional surgery,[2] "recurrence" of carcinoma versus new primary tumors[22, 23] and the paramount need for more frequent examination and closer observation of that particular organ or system in which patients have developed their initial primary cancer.

SUMMARY

A case report of 3 primary bronchogenic carcinomas arising in a left lower lobe is presented. Two of these neoplasms metastasized separately to regional nodes and distant organs. Problems of clinicopathologic interest and the theory of the multicentric origin of cancer evoked by the case are discussed briefly.

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showing juxtaposed histologic variants of bronchogenic carcinoma do not indicate whether each element metastasized separately[2, 3] as occurred in the present instance.

It touches, too, in microcosm, on many points found in some recent papers dealing with carcinoma of the lung including the re-emphasis on classification based on histology,[4, 5, 6] site of origin of pulmonary cancer correlated with histologic type,[7] the significance[4, 6-13] or lack of significance[5, 14, 15] of cell type, and the location of metastases compared with cell type.[16] In two excellent recent papers, Walter and Pryce[4, 7] have correlated the histologic type of pulmonary cancer with the site of origin in a large series of surgically resected and necropsy specimens. Their observations with regard to data gleaned from surgical rather than necropsy material are important, as is their insistence, along with Willis, on greater recognition of the secondary changes in tumors which obscure cell type and make comparisons of different authors' classifications difficult. These two papers[4, 7] are of particular value in helping to establish that disparate statements in the literature may be due to the use of necropsy material alone[5, 15] rather than surgical specimens used in concert with necropsy findings. For example, few people would agree with Olcott's impression of the clinical course and behavior of oat cell carcinomas of the lung derived from his 8 cases.[5] The general experience with oat cell carcinoma (also evidenced here by the extent of the metastases of this component) conforms fairly closely to the reports from the Mayo Clinic.[9] Survival beyond one year after surgical resection is a rare occurrence. It may be that patients in whom a diagnosis of oat cell carcinoma can be made by bronchoscopic biopsy should have no further operative intervention as implied by the findings from the Mayo Clinic.[9]

In recent years, renewed emphasis has been placed on the correlation of cell type with clinical course[7, 8-13, 17] although some have denied that it has any role in helping to determine prognosis and survival.[3, 14] It is becoming increasingly evident that there are many interrelated and interdependent factors (rather than any single one) which are important in improving operability, survival, and salvage rate for carcinoma of the lung. Among these factors (not only in carcinoma of the lung but at other sites as well) are histologic type, location and size of the primary tumor, presence or absence of vascular and lymphatic invasion, and the status of the margins of resection as well as a group of only dimly envisioned "host factors" (See Ref. 18).

Yet another facet of this case, apart from the presence of the 3 distinct primary carcinomas with separate lines of metastasis of 2 of the tumors, is the problem of multicentric versus unicentric origin of carcinoma of the lung. McGrath, Gall, and Kessler[2] used the variations in histologic type found when preparing large tissue sections of pulmonary tumors as one basis for support of the theory of the multicentric origin of bronchogenic carcinoma, but apparently none of their "mixed" cases metastasized as distinctive histologic entities. The striking illustrations by these same authors[2] of in situ carcinoma in bronchi adjacent to infiltrating tumor as well as multiple sites in bronchi well

EXPERIMENTAL PREVENTION OF VENTRICULAR FIBRILLATION FOLLOWING HYPOTHERMIA AND INDUCED CARDIAC ARREST

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GREAT progress has been made in the field of intracardiac surgery in the past few years. Among the factors making this possible has been the use of hypothermia to protect the cardiac and cerebral cells during periods of cessation of circulation.

Hypothermia, however, has certain inherent dangers which complicate its general application. The greatest of these is the increased susceptibility of the hypothermic heart to ventricular fibrillation. Several factors are prominent in this increased susceptibility to fibrillation and steps to prevent these are essential. It has been well shown that one of the greatest factors in the production of fibrillation is that of hypoventilation and a subsequent fall in the pH of the blood.[1, 15] It is also known that the potassium level falls consistently while the calcium level rises with a fall in the temperature.[3] Sodium and magnesium levels apparently remain constant.[2]

Hooker,[6] in 1930, showed that potassium chloride was effective in the treatment of ventricular fibrillation. However, Kyle and Kirby[7] have recently shown that potassium chloride is not as effective as electric shock, notwithstanding earlier reports that electric shock was ineffective during hypothermia.[10] They point out that a serious disadvantage to the use of potassium chloride is that cardiac arrest occurs in diastole as the result of myocardial depression and there is then difficulty in establishing a normal rhythm.

Although defibrillation can be accomplished by the use of potassium chloride or by electric shock, the prevention of ventricular fibrillation is the desired goal. Magnesium sulfate, quinidine, and the antihistamines were studied pursuant to this objective.

Magnesium has long been known to be a myocardial depressant through its action on delay of the A-V and ventricular conduction times[12, 13, 14] and, in sufficient concentration, is known to cause cardiac standstill.[11] Quinidine, also, is a myocardial depressant affecting general myocardial excitability as well as conduction and contractility. Its action has been studied in normothermic animals and it has been shown that 10 mg. per kilogram given intravenously will protect the myocardium from ventricular fibrillation induced by

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occurring immediately. During the period of occlusion of 6 to 7 minutes, the heart in all cases would gradually resume a slow bradycardia. During this period of occlusion the septum was explored through a right auricular incision. In 6 of the 9 animals, ventricular fibrillation occurred with the return of inflow of blood or when cardiac massage was used in an attempt to restore normal rhythm.

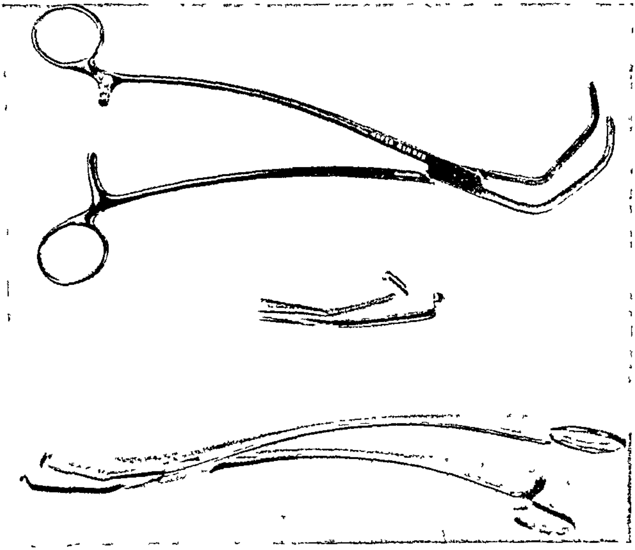

Fig. 1—The All-Purpose Vascular Clamp The clamp is useful in those situations where compression by a vertical device is more desirable than occlusion by the various horizontal hemostats now available The Atraugrip* feature keeps the clamp from slipping, and apparently does not injure the vessels. It has a shaft, including the handle, 22 cm long and its curve is away from the operator so that it may be held by an assistant without obstructing the surgeon's view

Acetylcholine with magnesium sulfate: In the second group (Table II), magnesium sulfate was used in addition to the 100 mg. of acetylcholine. This was administered by two different methods. In 2 dogs the same procedure as described above was carried out except that, prior to the injection of the acetylcholine, the coronary arteries were perfused with a solution of 2 Gm. of magnesium sulfate. This resulted in various degrees of heart block which reverted to a normal rhythm in 5 to 7 minutes. At this time the acetylcholine was injected with immediate and total standstill for the period of occlusion. In one of these, a normal rhythm was obtained after a short period of massage, in the other the heart stopped in diastole and ventricular fibrillation occurred with massage.

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*Patent by Pilling & Co, Philadelphia, Pa an electric shock, although fibrillation can still be produced by a stronger current or repeated shocks.[4, 7, 8, 10] Antihistamines have been shown to have an effect similar to quinidine in normothermic animals.[8]

EXPERIMENTAL WORK

Method.—Adult mongrel dogs were anesthetized with sodium pentobarbital (30 mg./Kg.) and cooled in an ice and water bath to a rectal temperature of 31° C. In all cases, the temperature would then fall to 27°-29° C. during the operation. After intubation and mechanical respiration, both chest cavities were opened through the fourth intercostal space and the sternum transected. The azygos vein was ligated and the superior and inferior venae cavae dissected and umbilical tapes passed around them. After opening the pericardial sac, the base of the aorta was dissected free and a tape passed around it. A tape was also passed through the transverse pericardial sinus for identification. (In later cases, we found that the operation could be done as easily through the right chest alone and that the animals tolerated the procedure much better when the sternum was left intact.)

After occlusion of the venae cavae for 45 seconds to allow the heart to empty, the aorta was occluded and various cardioplegic mixtures in 8 c.c. volume were injected proximal to the aortic occlusion and distal to the aortic valve cusps to ensure perfusion of the coronary arteries. A Berman* vascular clamp (Fig. 1) was then placed across the aorta and pulmonary artery through the transverse pericardial sinus. This not only prevents the backflow of the cardioplegic mixture from the coronary system, but prevents the possibility of coronary air embolism in cases of septal defects wherein the approach is through the right heart.

The experimental studies were divided into 4 groups according to the cardioplegic material used. The figures summarizing each of these groups express, under "Results," what occurred after the 5- to 7-minute period of induced cardiac arrest.

Acetylcholine: In the first group (Table I), 100 mg. of acetylcholine in 8 c.c. volume was perfused into the coronary system with standstill of the heart

TABLE I. ACETYLCHOLINE*

ANIMAL	RESULT
I	Ventricular fibrillation
II	Ventricular fibrillation
III	Ventricular fibrillation
IV	Ventricular fibrillation
V	Normal rhythm followed by ventricular fibrillation
VI	Ventricular fibrillation
VII	Ventricular fibrillation
VIII	Normal rhythm
IX	Ventricular fibrillation after 7 minutes of weak contractions

*In each case the procedure was carried out at a temperature of 28°-29° C. Acetylcholine, 100 mg. in 8 c.c. volume, was perfused through the coronary system to produce cardiac arrest.

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*Jacob K. Berman, M.D.

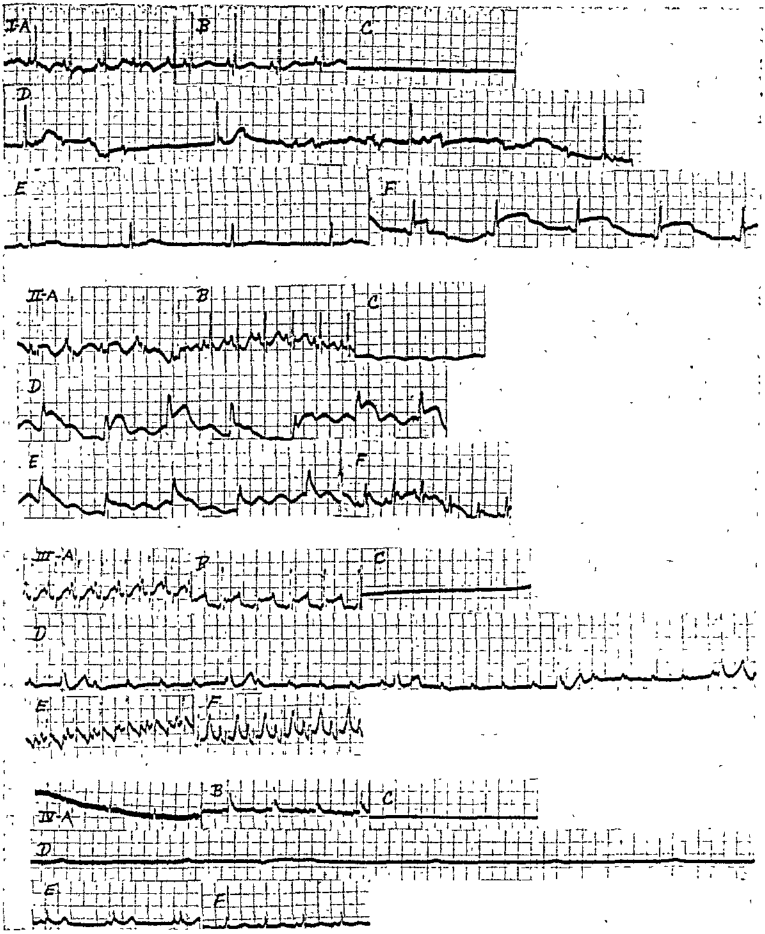

Fig. 2.—Lead II ECG tracings of quinidine animals I-IV. In each case, *A* is a preoperative, normal temperature tracing.

Animal I shows the effects of quinidine in *B*, a prolongation of Q-T interval and elevation of the T wave. Arrest (*C*) is followed by a complete heart block (*D*) which reverted spontaneously to a normal sinus rhythm (*E*). Eight minutes after restoration of circulation there was a normal sinus rhythm with a marked current of injury (*F*).

Animal II shows the same pattern in *A*, *B*, *C*. An idioventricular rhythm with a current of injury (*D*) was established after arrest. Atropine, gr. 1/150, was given 8 minutes later with the establishment of a normal sinus rhythm (*E*). Due to poor tone, Adrenalin 1:1000, 1 c.c., was given 10 minutes after the atropine (*F*).

Animal III shows the "injury potential" at the QRS-ST junction (*B*). Arrest (*C*) was followed by a complete heart block (*D*). This reverted to a normal sinus rhythm in one minute (*E*) and, in 15 minutes, the T waves had become upright.

Animal IV also shows the "injury potential" in *B*. Arrest (*C*) was followed by an idioventricular rhythm originating above the bifurcation with an auricular standstill (*D*). A normal sinus rhythm was established in 5 minutes (*E*). *F* shows the pattern 45 minutes later after the chest was closed.

TABLE II. ACETYLCHOLINE AND MAGNESIUM SULFATE*

ANIMAL	$MgSO_4$ (GM.)	HOW GIVEN	RESULTS
I	2.0	Given i.v. 7 minutes before acetylcholine	Normal rhythm followed by fibrillation in 25 min.
II	2.0	Mixed with acetylcholine	Fibrillation after 4 min. of massage
III	0.4	Mixed with acetylcholine	Fibrillation after 25 min. of massage
IV	0.25	Mixed with acetylcholine	Fibrillation after 1 min. of good beat
V	0.52	Mixed with acetylcholine	Fibrillation before release of inflow
VI	0.39	Mixed with acetylcholine	Normal rhythm
VII	1.0	Mixed with acetylcholine	Fibrillation after 4 min. of occlusion
VIII	2.0	Given i.v. 7 minutes before acetylcholine	Fibrillation with massage

*Acetylcholine, 100 mg. in 8 c.c. volume, perfused into the coronary system in each case to produce arrest.

In 6 other animals magnesium sulfate was perfused through the coronary system in a mixture with the acetylcholine, the amount varying from 0.25 Gm. to 2 Gm. In 4 cases, ventricular fibrillation occurred immediately upon return of circulation or before. In these animals, the hearts were defibrillated by the use of 2 to 3 c.c. of potassium chloride in a concentration of 1.33 mEq. per cubic centimeter perfused into the coronary system. This was followed by an equal volume of 5 per cent calcium gluconate. This was successful in each attempt although the procedure had to be repeated 3 or 4 times in some cases before a normal rhythm was obtained. In 2 of these hearts, there was a spontaneous reversion to fibrillation after a short period of relatively normal contractions.

Acetylcholine with quinidine: In the third group of animals (Table III), quinidine gluconate was given preoperatively in an attempt to prevent fibrillation. The same technique as described under "Method" was carried out except that the animal was given quinidine, 10 mg. per kilogram, intravenously before cooling was started. The quinidine was diluted to 10 c.c. volume with normal saline and was administered over a period of approximately 10 minutes.

TABLE III. ACETYLCHOLINE AND QUINIDINE GLUCONATE*

ANIMAL	RESULTS
I	Spontaneous normal rhythm
II	Spontaneous normal rhythm
III	Spontaneous normal rhythm
IV	Spontaneous normal rhythm
V	Ventricular fibrillation after Adrenalin
VI	Spontaneous normal rhythm
VII	Spontaneous normal rhythm
VIII	Spontaneous normal rhythm

*Quinidine, 10 mg. per kilogram, was given intravenously immediately prior to cooling in each case. Acetylcholine, 100 mg. in 8 c.c. volume, was used to produce arrest.

The animal was then cooled and the procedure completed using acetylcholine for cardiac arrest, which was maintained from 5 to 7 minutes. In each case, the heart maintained good tone during this period of arrest and in all but one animal resumption of normal rhythm was immediate upon the release of inflow

In this group, the right ventricular wall was incised in 4 cases as a stimulus for fibrillation. However, fibrillation did not occur. In 5 of these cases atrophine gr 1/150 in 4 c.c. volume was perfused into the coronary system before resumption of inflow. This seemed to give a slightly quicker return to normal sinus rhythm. The animals in this group were observed from one day to one month postoperatively with return of normal ECG tracings and grossly normal behavior in all respects.

Figs. 2 and 3 show the ECG tracings of this group of animals except for the case which ended in ventricular fibrillation.

Acetylcholine with Histadyl. In a fourth group of animals, the same technique, as described under "Method," was carried out except that the antihistamine, thenylpyramine (Histadyl Lilly), was used in conjunction with the acetylcholine (Table IV).

TABLE IV. ACETYLCHOLINE AND HISTADYL*

ANIMAL	HISTADYL (MG./KG.)	HOW GIVEN	RESULTS
I	5.0	Intravenously prior to acetylcholine	Normal rhythm
II	5.0	Mixed with acetylcholine	Fibrillation
III	2.5	Mixed with acetylcholine	Normal rhythm

*Acetylcholine 100 mg./Kg. in 8 c.c. volume used to produce arrest by perfusion of coronary system.

A normal rhythm was established following standstill in 2 cases. In one of these, 2.5 mg. per kilogram of Histadyl was used in a mixture with 100 mg. of acetylcholine perfused into the coronary system. In the other case, 5 mg. per kilogram of Histadyl was injected into the inferior vena cava immediately prior to the administration of acetylcholine into the coronary system.

In the third case, 5 mg. per kilogram of Histadyl plus 100 mg. of acetylcholine in the coronary system resulted in a complete flaccid standstill which would not revert spontaneously to good contractions with the release of inflow. Massage was required to support the circulation and this precipitated fibrillation. In the 2 cases where a normal beat was obtained the heart tone was not as good as with the use of quinidine. Also standstill was not as complete and resumption of beats was slower than in the group subjected to quinidine.

DISCUSSION

Ventricular fibrillation is one of the chief complications occurring during hypothermia. In intracardiac surgery with the use of cardiac standstill, this occurs most frequently when manual massage is required to restore the beat. This is due to the mechanical stimulation of the myocardium as well as to the change in pH and electrolytes. For this reason we felt that if we could protect the myocardium by a depressant we could help overcome this complication.

The effects of quinidine were studied in 9 dogs under hypothermia and cardiac arrest and compared with a series of 9 animals under similar arrest with acetylcholine alone, with a group of 8 animals in which depression of the myocardium was attempted with magnesium sulfate and with a group of 3 animals in which Histadyl was used.

to the heart and without massage. In one animal, although the heart resumed beating spontaneously after the relase of tourniquets and the clamp, the beats were weak, so one cubic centimeter of 1:1000 Adrenalin was given intravenously whereupon ventricular fibrillation occurred.

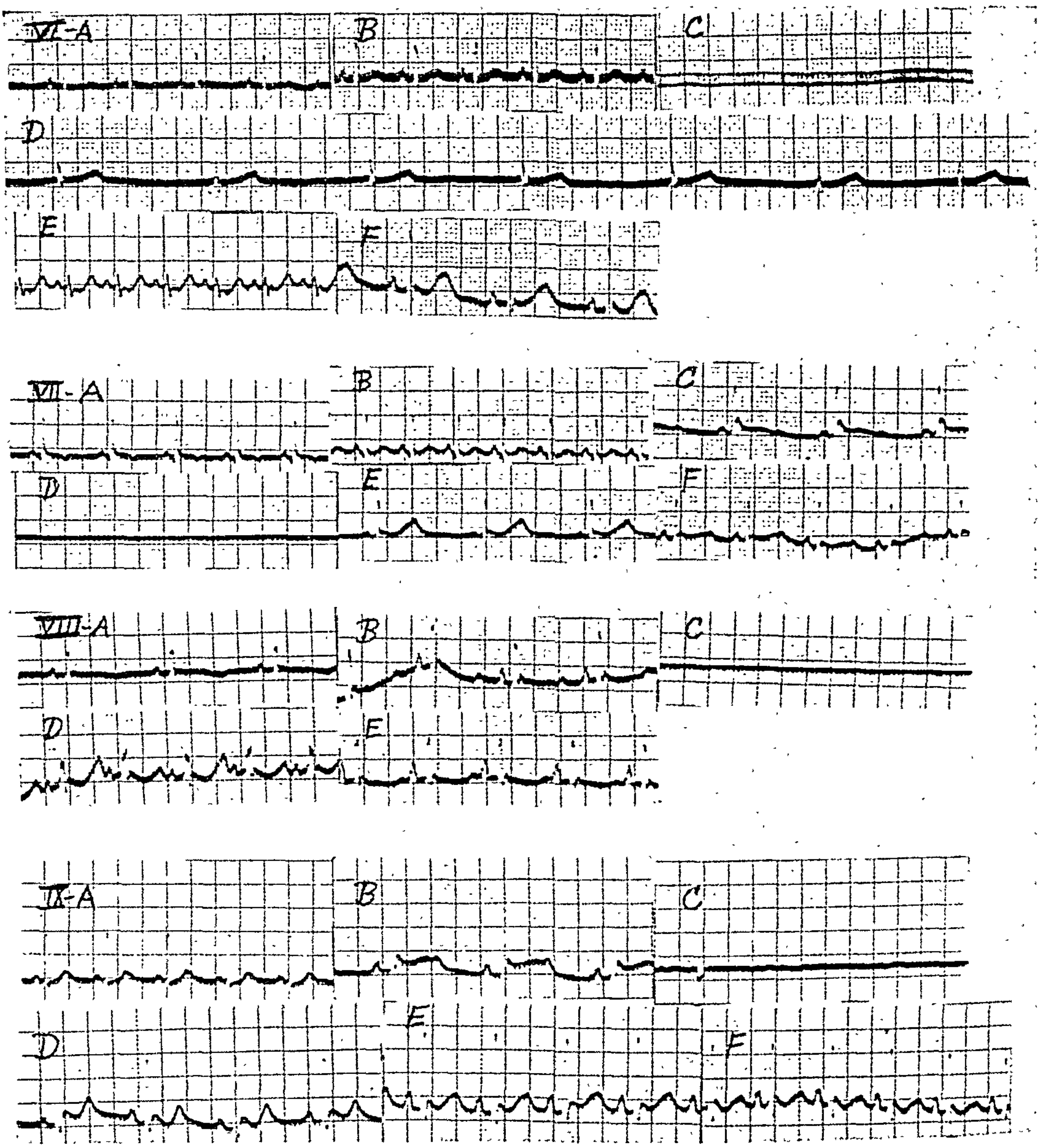

Fig. 3.—Lead II ECG tracings of quinidine animals VI-IX.

Animal VI shows the effects of quinidine (*B*) followed by arrest (*C*). A normal sinus rhythm was established immediately upon restoration of circulation (*D*). *E* shows the effects of atropine and 45 minutes later after these had disappeared (*F*).

Animal VII shows the quinidine effect (*B*) and the "injury potential" at 27° C. (*C*). Arrest (*D*) was followed by a normal sinus rhythm (*E*). *F* shows the pattern one hour later after the chest was closed and the animal warmed to 32° C.

Animal VIII shows the "injury potential" at 28° C. (*B*). Arrest (*C*) was followed by a normal sinus rhythm (*D*). *E* shows the tracing 11 minutes after restoration of circulation.

Animal IX shows the "injury potential" at 29° C. (*B*). Arrest (*C*) was followed by a normal sinus rhythm (*D*). *E* shows the pattern 12 minutes later. *F* is the tracing after incision and repair of the right ventricular wall.

with the use of magnesium, but that the heart was protected by quinidine and Histadyl Of these 2 drugs, quinidine appears to be superior although Histadyl was not used extensively and deserves further study Quinidine decreases the complications of hypothermia

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We have found, contrary to the theory that the magnesium ion is a cardiac depressant, that under hypothermia the irritability of the heart seems to be increased by the use of the magnesium and that it is not protective against ventricular fibrillation. With the use of magnesium, fibrillation occurred much sooner and with less stimulation than in the acetylcholine group.

In most cases, fibrillation can be stopped by the use of potassium chloride and calcium gluconate injected in the coronary system. Good cardiac rhythm can usually be established although this is sometimes done with difficulty and a reversion to fibrillation is common.

In the use of quinidine, one of the main advantages is that arrest in diastole does not occur and the heart resumes regular rhythm without the use of manual massage which is one of the chief causes of fibrillation. Cardiac arrest is produced sooner and total arrest is maintained longer than with the use of acetytcholine alone. This conservation of cardiac energy during occlusion is a factor in decreasing fibrillation. When the factor of cardiotomy is added to those of hypothermia and arrest, quinidine continues to be protective.

Although the work with the antihistamines has not been extensive enough for any conclusions, in the 3 cases where it was used the hearts did not respond as well as with the use of quinidine. The standstill was less complete and restoration of normal rhythm was slower than with the use of quinidine.

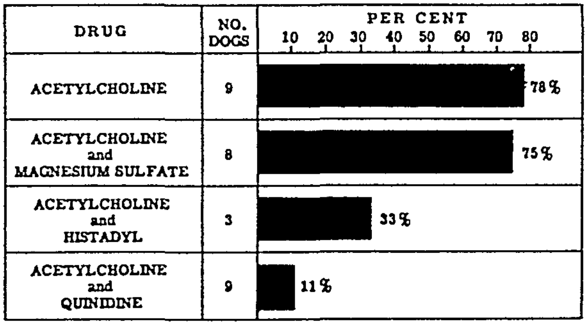

Fig. 4.—Percentage of dogs in which fibrillation occurred subsequent to hypothermia and cardiac arrest induced by acetylcholine alone and acetylcholine used in combination with magnesium sulfate, Histadyl, and quinidine.

Fig. 4 shows the percentage of dogs in which fibrillation occurred subsequent to hypothermia and cardiac arrest in each of the 4 groups of animals we have studied.

ECG tracings were taken of all animals in each group throughout the entire procedure. However, in the interest of brevity and the elimination of repetition, only the tracings of the quinidine group are shown.

SUMMARY

Cardiac arrest was produced in hypothermic dogs with acetylcholine alone and in combination with magnesium sulfate, quinidine, and Histadyl. It was found that the incidence of ventricular fibrillation was not decreased posterior aspect of the mural leaflet are engaged and avulsed. Upon completion of this procedure, a major regurgitant jet can be palpated in the region of the posterior commissure, thus simulating clinical mitral insufficiency.

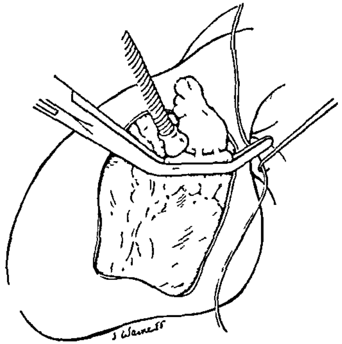

Fig 1.—A rectangular segment of pericardium is excised for preparation of the graft A purse-string suture is placed around the base of the left auricular appendage, and a Satinsky clamp is placed distal to it.

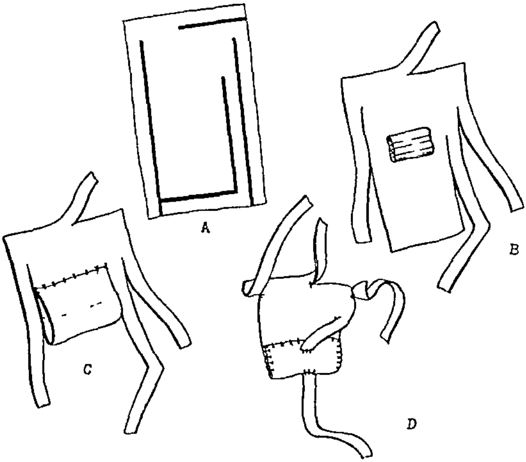

Fig 2.—A, A rectangular segment of pericardium measuring approximately 4 by 7 cm is secured, and tails are prepared

B and *C*, A piece of muscle is incorporated into the valve graft to provide the bulk and substance necessary to tamponade the regurgitant jet.

D, Upon completion, a valve graft is created which can be anchored at multiple points along its base, as well as at its apex

Technique of Repair of Mitral Insufficiency.—A pericardi [illegible] gr [illegible] first prepared for insertion (Fig. 2). A rectangular segme [illegible]

THE SURGICAL CORRECTION OF MITRAL INSUFFICIENCY BY USE OF A PERICARDIAL VALVE GRAFT

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A SIMPLE closed procedure has been developed to abolish a regurgitant lesion at the posterior commissure without impairing the functional mobility of the aortic leaflet. This has been accomplished effectively by the addition of a pericardial valve graft to the posterior half of the mural leaflet of the mitral valve.

METHODS

Mongrel dogs, weighing from 20 to 25 Kg., were anesthetized with intravenous Pentothal sodium, after which a left thoracotomy incision was performed through the fifth intercostal space. Mitral incompetence was produced by resecting the posterior portion of the mural leaflet with a mastoid rongeur, after which the chordae in this region were avulsed with a nerve hook.

Initially, an attempt was made to prepare a series of animals with severe chronic mitral insufficiency for future surgical correction. However, production of a major mitral regurgitation was invariably attended by a mortality due to pulmonary edema within 12 to 24 hours. Only insignificant lesions could be produced with survival.

The necessity for the immediate correction of the severe regurgitant lesion became apparent. The present experiment, therefore, comprises a series of animals in which the creation of a significant mitral insufficiency was followed by the immediate insertion of a pericardial valve graft for its correction. Intracardiac pressures from the left atrium and aorta, and an electrocardiogram were obtained before and after the creation of the mitral regurgitation, and again after its surgical correction.

Technique of Creation of Mitral Insufficiency.—The pericardium is opened longitudinally, and a purse-string suture is placed around the base of the left auricular appendage. A Satinsky clamp is placed distal to it, after which the appendage is opened and irrigated with saline (Fig. 1). The bare left index finger is inserted into the atrium, and the mitral valve area is explored. The posterior portion of the mural leaflet is resected with a mastoid rongeur passed transauricularly along the palpating finger. The rongeur is removed, and a nerve hook is similarly introduced. The chordae inserting into the residual

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located. The first suture* is placed to the right of the posterior descending artery, into the left ventricle just beneath the posterior commissure. The suture then passes between the valve leaflets into the left atrium, and out through the auricular appendage. The end of the suture is immediately attached to the corresponding tail of the valve graft. The next suture is placed in the mural leaflet anterior to the regurgitant jet at a point approximately midway between the two commissures. This suture is passed from outside the heart just below the coronary vessels, and is immediately angled upward to enter the left atrium at the junction of the valve leaflet with the annulus. This suture, too, is attached to the corresponding tail of the graft. A third suture is similarly placed midway between the first two (Fig. 3). The finger is then placed into the left ventricle against the posterior papillary muscle. A suture is passed into the left ventricle at this site, and is guided between the valve leaflets into the atrium (Fig. 4). It is withdrawn from the auricular appendage, and attached to the tail of the valve graft (Fig. 5). The pericardial valve graft is inserted into the atrium and is snugged into its proper position by pulling on the appropriate sutures. The tails are then anchored to the ventricular myocardium (Figs. 6 and 7, *A* and *B*).

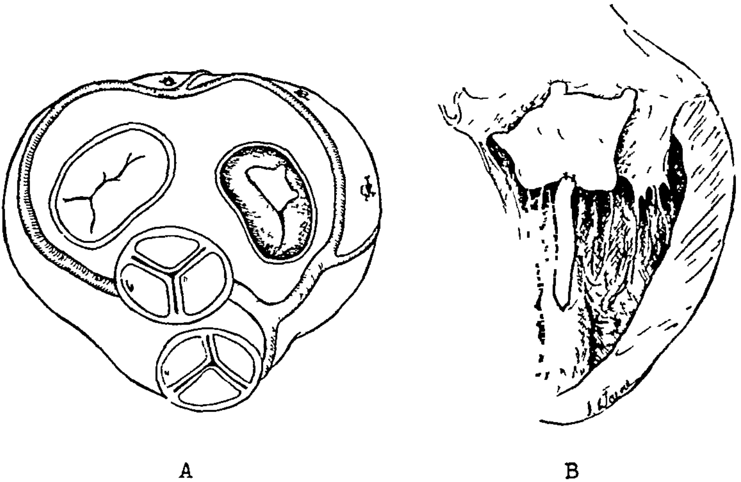

Fig. 7.—*A*. The valve graft is snugged into position so that the regurgitant area is tamponaded.

B. Same with heart open, demonstrating the final placement of the tails of the graft into the valve leaflet and papillary muscle.

In this fashion, a valve graft placed across the posterior portion of the mitral orifice can replace a portion of a resected leaflet of the mitral valve. The bulkiness of the graft immediately tamponades the regurgitant jet without in any way interfering with the mobility of the aortic leaflet.

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*Prepared by J. A. Deknatel & Son, Inc., Queens Village, Long Island, N. Y.

measuring approximately 4 by 7 cm., is secured. Tails are prepared so that the completed valve may be anchored along both its apex and base (Fig. 2, *A*). A piece of muscle is incorporated into the valve graft to provide the bulk and substance necessary to tamponade the regurgitant jet (Fig. 2, *B* and *C*). Upon completion, a valve graft is created which can be anchored at multiple points along its base, as well as at its apex (Fig. 2, *D*). The anchorage of the apex into the posterior papillary muscle serves as a chorda tendinea. The bare left index finger is again inserted into the atrium, and the posterior commissure is

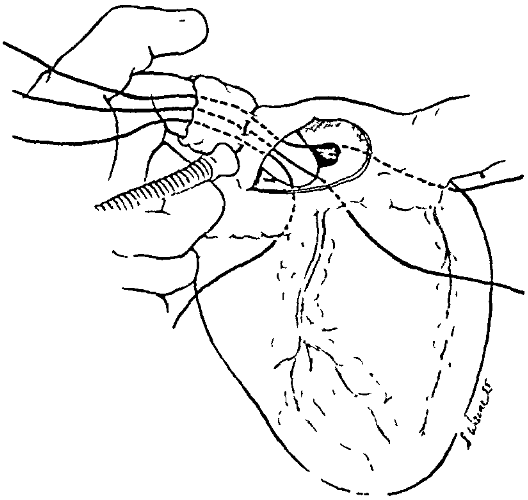


Fig. 3.

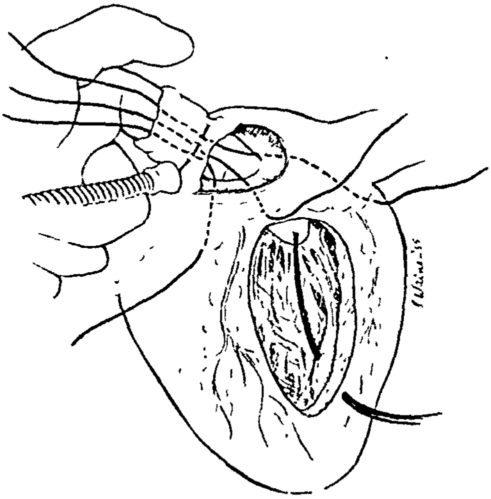


Fig. 4.

Fig. 3.—The first suture is placed posteriorly into the left ventricle just beneath the posterior commissure. It is then passed between the valve leaflets into the atrium, and out through the auricular appendage. The second suture is placed anterior to the regurgitant jet, from below the coronary vessels exteriorly, to the junction of the leaflet with the annulus internally. The third suture is similarly placed midway between the first two.

Fig. 4.—The suture is passed into the posterior papillary muscle for insertion of the graft chordae.

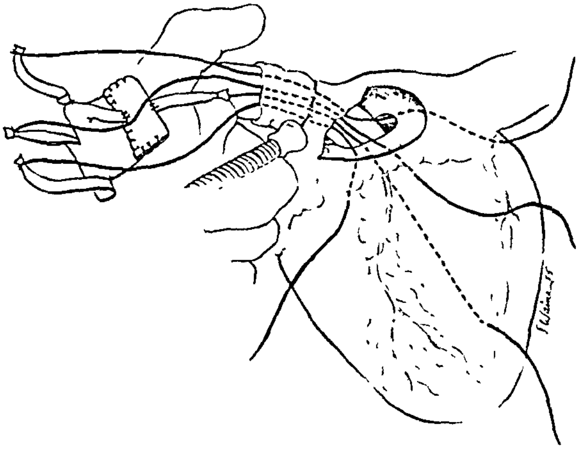


Fig. 5.

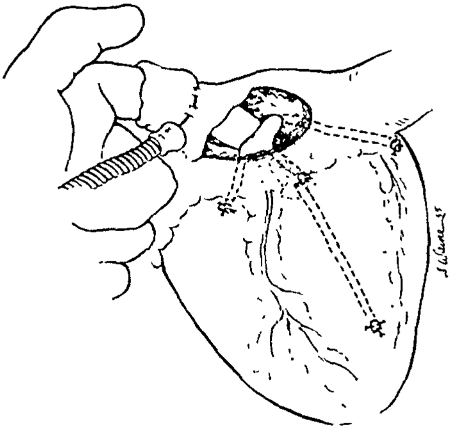
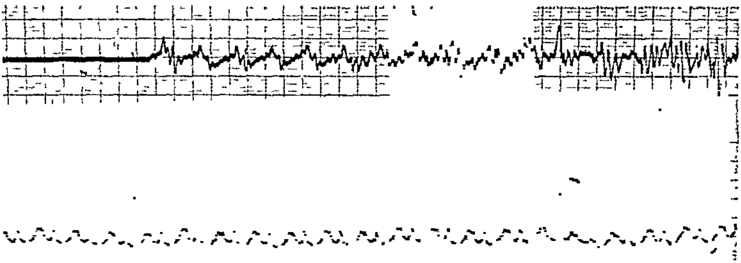


Fig. 6.

Fig. 5.—The sutures are attached to the respective tails of the graft.

Fig. 6.—The pericardial graft is inserted into the atrium and is snugged into its proper position by pulling on the appropriate sutures.



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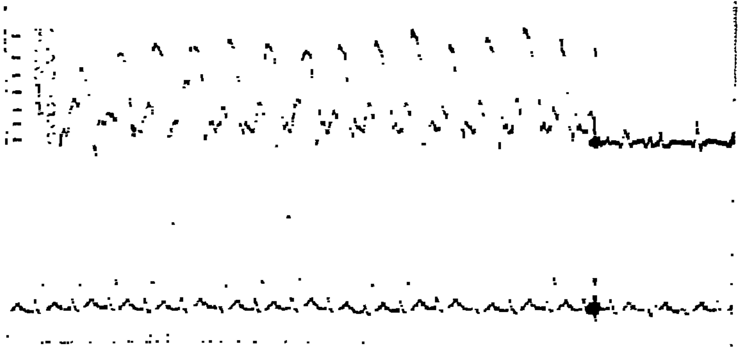

Fig.

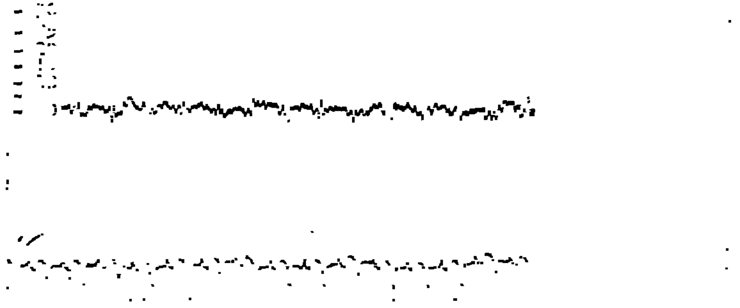

Fig 1

Fig. 8.—Normal left atrial pressure tracing taken prior to resection of the mural leaflet of the mitral valve.

Fig. 9.—Same, after production of mitral insufficiency by resection of the posterior portion of the mural leaflet. Note marked elevation of C wave.

Fig. 10.—Same, after insertion of pericardial valve graft. Note complete reversion of pressure tracing to normal.

RESULTS

1. *Period of Instruction.*—The first series of animals was prepared during the development of the techniques described, with no attempt at survival. Initially, the need for bulk in the graft was not appreciated and, in the first 4 animals, a pericardial valve graft alone, without enclosed muscle, was inserted. The regurgitant lesion remained uncontrolled, and pulmonary edema ensued in all. The placement of the posterior suture proved to be the next troublesome factor. It seemed that regardless of how far posteriorly this suture was placed, the most posterior portion of the mitral orifice could not be tamponaded. This was finally solved by passing the suture to the right of the posterior descending artery, so that it was actually passed into the left ventricle from the external aspect of the right ventricle, through the interventricular septum. Several animals were lost initially, because the necessity for a ventricular tail to maintain the position of the graft was unrecognized. With each ventricular systole, the graft was herniated into the atrium without controlling the regurgitation.

Finally, a group of 6 animals were prepared. Two were sacrificed at 6 months, and 3 at 8 months. One died at 5 months of embolization.

2. *Physiologic Observations.*—In all animals (Table I), after insufficiency was produced, a typical regurgitant atrial pressure tracing was observed. There was an elevation of the atrial C wave (Fig. 9). After the valve graft was inserted, the regurgitant jet was abolished, with reversion of the atrial pressure tracing to normal (Figs. 8, 10).

TABLE I. LEFT HEART PRESSURE AT THORACOTOMY BEFORE AND AFTER INSERTION OF GRAFT

DOG NO.	LEFT ATRIUM (MM. HG)	PEAK OF C-V WAVE	AORTA	COMMENTS
No. 4 Initial	5-10	10		Complete obliteration of regurgitant jet after insertion of graft
After regurg.	10-35	37		
After correction	0-5	5		
No. 2 Initial	7-10	10		Good control of regurgitant jet
After regurg.	10-30	30		
After correction	3-7	7		
No. 3 Initial	6-10	10		Good correction of MI with valve graft; minimal residual jet palpable at posterior aspect of mitral orifice
After regurg.	10-30	30		
After correction	5-10	10		
No. 1 Initial	5-8	8		Regurgitation well controlled by valve graft; minimal residual jet palpable
After regurg.	5-25	25		
After correction	0-5	5		
No. 6A Initial	5-10	8	120/50	Mitral regurgitation poorly repaired; a large jet was palpable postgraft; the graft was too small and not bulky enough; animal died in 24 hours
After regurg.	5-20	20	100/44	
After correction	5-20	20	90/40	

3. *Pathologic Observations.*—In 4 animals, 2 of which were sacrificed at 6 and 8 months, respectively, no regurgitation existed at time of sacrifice. These

two categories, the regurgitant jet is found posteriorly emerging from the region of the posterior commissure. Invariably, valvular competence is maintained anteriorly.

Bailey[17] believes that normally, although there is a considerable excess of valve leaflet for coaptation anteriorly, there is very little such excess tissue posteriorly (Fig. 12). This relationship is seriously altered by the thickening, rolling, and shortening of the valve leaflets and chordae, caused by the rheumatic infection. The anterior portions of both the mural and septal leaflets, having an excess of valve tissue, seem to tolerate this shortening without loss of competence. The posterior portions of these leaflets, however, barely coapting in the normal state, are rendered incompetent by this pathologic process. In truth, there seems to be an actual deficiency of valvular substance of the posterior aspect of the mural leaflet, which may be so severe that only a residual rim of atrophic tissue remains. The physiologic sequelae of the regurgitant lesion revolve about the increased burden placed upon the left ventricle, which becomes dilated and hypertrophied. This is then followed by a secondary dilatation of the mitral annulus.

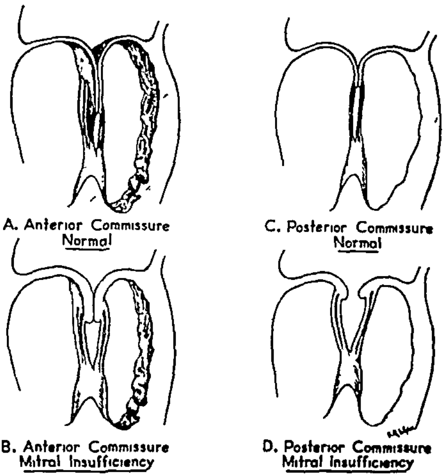

Fig. 12.—The regurgitant jet invariably emerges through the posterior commissure. Note the apparent deficiency in leaflet of the posterior portion of the mural leaflet. Compare to Fig. 21 postoperatively.

It would seem, therefore, that the surgical correction of mitral insufficiency must be directed toward the addition of substance to the posterior portion of the deficient mural leaflet. This valve substance must tamponade the regurgitant flow of blood by enabling the posterior portion of the aortic leaflet to coapt animals had spent an active postoperative kennel existence. In each, the graft had become adherent to the mitral valve, and completely replaced the resected portion of the leaflet. The base of the graft was entirely adherent to the annular attachment of the valve. The apical chorda was thickened and fibrotic. The graft seemed to have completely and effectively replaced the resected portion of the valve (Fig. 11).

In one animal sacrificed after 6 months, although the major regurgitant jet had been abolished, a residual fine jet could still be palpated. In this animal the valve graft was found to be in proper position, but the base was adherent only at its points of fixation. The regurgitant jet, palpated prior to sacrifice, had emerged between the points of fixation. This finding led to the fixing of the base of the graft at four points in the ensuing experiments.

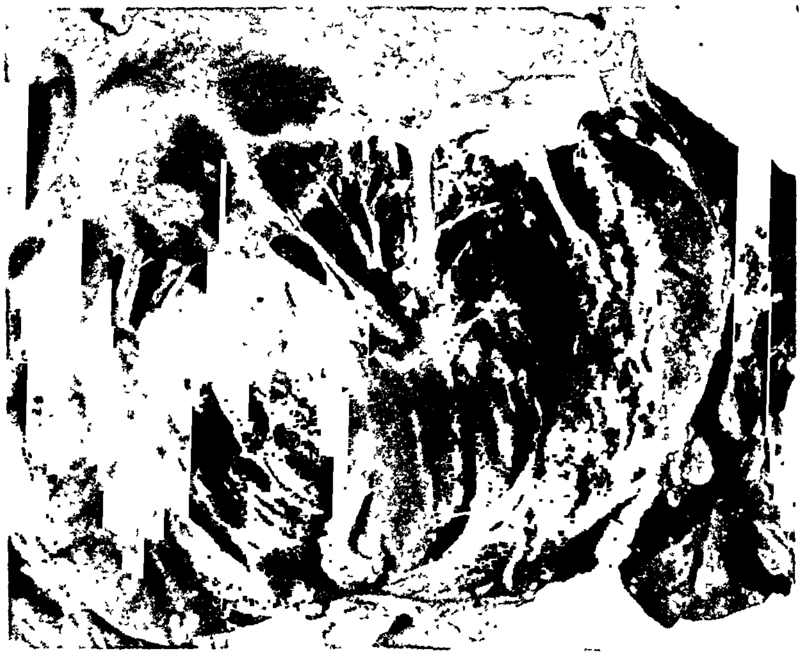

Fig. 11.—Same dog, 8 months postoperative. The graft has completely replaced the resected portion of the mitral valve leaflet. The base is entirely adherent to the annular attachment of the valve. The apical chorda is thickened and fibrotic.

One animal died after 5 months, of peripheral embolization, including a terminal saddle embolus. Post-mortem examination revealed the base of the graft to have been avulsed at two points of fixation. The pericardial flap was found in the ventricular chamber, fixed at its apical portion, and surrounded by old and recent thrombus. This thrombus was the obvious source of the peripheral emboli.

DISCUSSION

Mitral insufficiency may vary in character as well as in degree. It may be a minor lesion associated with a predominant stenosis, the orifice of which is maintained by a thickened fibrous, or calcific ring. It may be considerably advanced, but still associated with moderate stenosis at the anterior commissure, or it may be the sole and major lesion of the mitral valve. In the latter seems to ignore the basic anatomic and physiologic hemodynamic aberration seen in mitral regurgitation. The third type of surgical attack revolves about a recognition of the primary anatomic deficiency in mitral insufficiency, namely that of an absolute loss of valve substance.

During cardiotomy for predominant mitral stenosis associated with insufficiency, it has been recognized that the regurgitant jet almost invariably emerges through the posterior commissure (Fig. 13). Proper placement of the exploring finger in this region can effectively tamponade the regurgitant jet as evidenced by a rise in systemic pressure and an increase in the fullness of the peripheral pulse. This fact, coupled with the pathologic finding of an absolute deficiency of valvular substance of the posterior portion of the mural leaflet, has led to the concept of tamponading the regurgitation by the proper placement of a valve graft which would adhere to, and add substance to, the atrophic portion of the mural leaflet.

Experience has demonstrated that the mobility of the aortic or septal leaflet cannot be impaired. This fact has led to the inevitable development of a valve graft positioned to add substance to the posterior portion of the mural leaflet, and thus effectively tamponade the regurgitant jet. The operation for the insertion of the valve graft is simple, and permits the utilization of a closed technique. The diagonal placement of the graft prevents the future development of a stenosis of the orifice by, at all times, ensuring full mobility of the aortic leaflet. In all animals, the graft is incorporated into the substance of the mural leaflet.

Undoubtedly, with the advent of open heart surgery, it will be possible to position this valve graft with even more accuracy.

SUMMARY

A closed technique is described for the placement of a pericardial valve graft into the orifice of the mitral valve for the control of mitral insufficiency.

The valve tamponades the regurgitant jet in the region of the posterior commissure, and adds substance to the deficient portion of the posterior aspect of the mural leaflet.

The valve is well tolerated, and has been incorporated into the substance of the mural leaflet.

The results of this experiment justifies the use of this technique in patients with regurgitant lesions of the mitral valve.

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against it during ventricular systole. At the same time, it has become obvious that the mobility of the aortic leaflet cannot be impaired without lethal consequences.

There have been many previous attempts at the surgical correction of mitral insufficiency, starting with Murray, Wilkinson, and McKenzie,[1] who first attempted to replace damaged cusps with vein grafts in 1938. Templeton and Gibbon,[2] in 1949, next attempted to replace segments of the tricuspid valve with venous or pericardial grafts, using an open right atriotomy. Bailey and co-workers[3, 4] attempted to occlude the regurgitant mitral orifice using a transventricular pericardial sling intended to act as an intermittent ball valve during ventricular systole. Johns and Blalock[5] reported this technique, but used a polyvinyl sponge prosthesis. Bailey and colleagues[6] next attempted to suture the posterior aspects of the mitral leaflets together. This technique must have immobilized the aortic leaflet. Harken[7] and Carter[8] and their associates inserted plastic prostheses into the ventricular chamber in an attempt to control the regurgitation in flutter-valve fashion. Glenn and co-workers[9] attempted to tamponade the regurgitant jet with a transchamber, vertically suspended, intracardiac graft. Davila,[10] on the other hand, has attempted to approach the problem from the reverse aspect of diminishing the size of the mitral orifice by use of a circumferential suture.

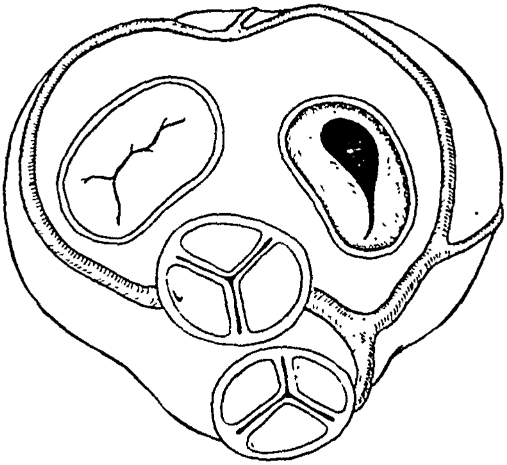

Fig. 13.—*A*, Normally, there is an excess of valve tissue for coaptation anteriorly.

B, Rheumatic valvulitis is accepted anteriorly without regurgitation because of the excess of substance.

C, Posteriorly, the valves barely coapt, and there is little overlap of tissue.

D, Posteriorly, however, where there is little room for leeway, loss of valve substance leads to failure of coaptation of the leaflets.

It seems that the procedures devised fit into three categories. The first revolves around the development of a transventricular flutter-valve designed to occlude (intermittently) the regurgitant portion of the mitral orifice. The valves inserted have been pericardial or prosthetic. The pericardial grafts become thickened, fibrotic, and attenuated.[11] The foreign bodies become fibrotic and, in a short period, both become immobile and useless. The second consists of an attempt to diminish the circumference of the mitral orifice. This method

BENIGN PEDUNCULATED, INTRALUMINAL TUMORS OF THE ESOPHAGUS

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POLYPOID, intraluminal tumors were among the first benign esophageal neoplasms to be described. Despite their infrequent appearance they are of considerable interest because of the sensational manner in which they may appear (by regurgitation of the growth) and because, if untreated, they may result in death of the patient. Yet they are amenable to curative surgical removal.

According to Harrington,[3] Sussins is credited with one of the first necropsy descriptions of an esophageal polyp (1559). In 1871, Coats described the necropsy findings in a patient who died with esophageal obstruction secondary to what he called an "intraluminal polypoid myxoma." He noted the benign nature of the lesion, emphasized its narrow band of attachment, and speculated about a possible cure of such a condition by surgical removal. The fact that these intraluminal esophageal tumors have been diagnosed infrequently is illustrated by a review by Totten and associates,[7] in 1953, wherein it is noted that they were able to find only 40 such cases recorded in the literature. The surprising appearance of the regurgitated tumor in the mouth of the patient occasionally facilitated the diagnosis. Successful surgical removal was first recorded in the American literature by Vinson[8] in 1922. The patient was the first of 6 who have been seen and treated by operation at the Mayo Clinic. With the exception of 2 cases, all have been reported previously in the literature.[3, 4] The pertinent clinical data in the 6 cases are summarized in Table I. Reports of the 2 most recent cases of these unusual esophageal tumors encountered at the Clinic follow.

REPORT OF CASES

Case 1.—A 56 year old man was first seen at the Clinic in 1951, at which time he complained of dysphagia of 6 months' duration with associated anorexia and loss of 48 pounds. X-ray examinations conducted by his home physician had revealed dilation of the esophagus, and esophagoscopic examination had given negative results. Gradually the patient had experienced intermittent regurgitation and increased substernal distress on swallowing. Dysphagia became so severe that he had had to be fed by tube for 2 weeks. When the tube was removed the patient had little trouble with swallowing for 6 weeks, but the symptoms of dysphagia, substernal fullness, regurgitation, and weakness returned.

The patient's medical history and a review of symptoms contributed nothing of significance.

From the Mayo Clinic and Mayo Foundation.
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Physical examination revealed evidence of much loss of weight and mild edema of the lower extremities, but otherwise no abnormalities were noted.

Laboratory studies disclosed a concentration of hemoglobin of 11.4 Gm. per 100 c.c. of blood and leukocytes numbered 13,500 per cubic millimeter of blood with some shift to the left noted on the differential blood smear. Urinalysis gave normal results. The sedimentation rate of erythrocytes was 53 mm. in one hour (Westergren method). X-ray examination of the thorax did not reveal any abnormality. By means of contrast medium, the roentgenologist observed evidence of a large intraluminal mass in the mid portion of the esophagus (Fig. 1). At the time of esophagoscopic examination, a pedunculated tumor was noted arising just below the esophageal introitus from the posterior and right lateral walls of the esophagus The tumor was lobulated and extended down into the lower third of the esophagus. It was the esophagoscopist's opinion that this was a large, pedunculated, intraluminal fibrolipoma.

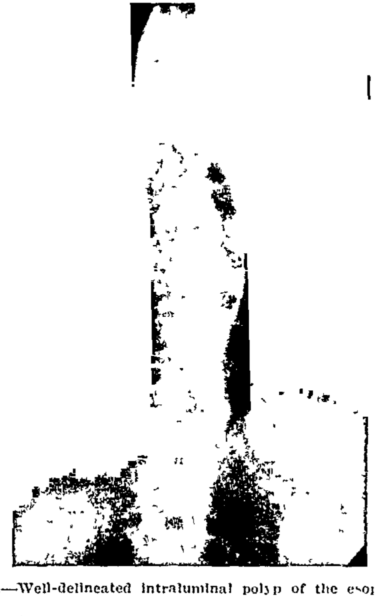

Fig 1.—Well-delineated intraluminal polyp of the esophagus

A cervical approach was used and the lesion, which measured 11 by 2 by 1.5 cm., was excised (Fig. 2). The pathologist reported the tumor to be a fibrolipoma (Fig. 3). The postoperative course of this patient was uneventful and follow-up studies showed that 6 years later he continued to be free of symptoms.

CASE 2.—A 24-year old woman was first seen at the Clinic in March, 1955, at which time she complained of progressive dysphagia of 13 months' duration. Initially, in February, 1954, she had noted only slight difficulty in swallowing solid food which was minimized by washing the food down with liquids. In a few months, however, the dysphagia had increased and she had sought the advice of a physician on two occasions. Without benefit of x-ray examination, the patient was told that her symptoms probably were due to increased "nervous

TABLE I. SUMMARY OF CLINICAL DATA IN SIX CASES

PERTINENT DATA	CASE NUMBER					
	1	2	3	4	5	6
Age; year encountered	62 yr. (1921)	24 yr. (1940)	42 yr. (1943)	21 yr. (1944)	56 yr. (1951)	25 yr. (1955)
Sex	Male	Male	Male	Male	Male	Female
Chief complaint	Coughed up growth	Sore throat	Regurgitation	Dysphagia	Dysphagia	Dysphagia
Duration	6 yr.	3½ yr.	8 yr.	10 yr.	6 mo.	1 yr.
Symptoms:						
Dysphagia	No	Yes	No	Yes	Yes	Yes
Vomiting	Yes	No	Yes	Yes	Yes	Yes
Pain	No	Ache beneath lower part of sternum on swallowing	Substernal pressure	Substernal, on swallowing	Substernal fullness	Substernal pressure
Loss of weight	30 pounds	12 pounds	30 pounds	20 pounds	48 pounds	19 pounds
Respiratory	No	Productive cough	Dyspnea, pneumonia	No	No	No
Melena	No	Yes	No	No	No	No
History of regurgitated tumor	Yes, twice	Yes, 4 times	Yes, once	Yes, many times	No	No
X-ray diagnosis		Cardiospasm	Cardiospasm	Intraluminal polypoid tumor	Large intraluminal mass	Cardiospasm with large intraluminal foreign body
Esophagoscopic finding	Intraluminal polyp	Intraluminal polyp	Intraluminal polyp	Intraluminal polyp	Intraluminal polyp	Intraluminal polyp
Therapy	Excision	Excision	Excision	Excision	Excision	Excision
Route of excision	Cervical	Cervical	Right transpleural	Oral	Cervical	Right transpleural
Level of tumor attachment	Just below introitus	Just below introitus	Just below introitus	Just below introitus	Just below introitus	Level of azygos vein
Postoperative complications	None	Paralysis left vocal cord	Empyema	None	None	Micrococcal enteritis
Result	Died in 6 months of abdominal carcinomatosis	Well	Well at last report	Well	Well	Persistent dysphagia months later; well at last report

tension.'' Soon she began to regurgitate much of the food she swallowed, so that by February, 1955, her diet was limited to liquids. Medical aid was sought again and x-ray studies of her esophagus, as well as an esophagoscopic examination, were done with the resulting diagnosis of cardiospasm. Dilation did not provide relief of her symptoms and she began to experience substernal fullness and aching when she swallowed. She had lost 10 pounds in 3 months.

The medical history and a review of symptoms contributed nothing, with the exception of some emotional problems that were of sufficient severity to require consultation with a psychiatrist.

Physical examination at the Clinic revealed a tense, pale young woman who had obviously lost some weight, but no other abnormality was noted.

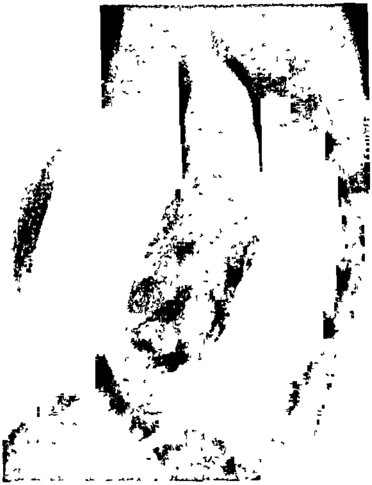

Fig. 4.—Note the marked dilation of esophagus associated with this intraluminal polyp.

Routine laboratory studies disclosed a concentration of hemoglobin of 11.7 Gm. per 100 c.c. of blood; erythrocytes numbered 3,720,000 and leukocytes 6,100 per cubic millimeter of blood with a normal differential count. The sedimentation rate of erythrocytes was 60 mm. in one hour (Westergren). Serologic tests and routine urinalysis gave negative results. X-ray examination of the thorax revealed the lung fields to be clear. Abnormality of the chest wall (pectus excavatum) was noted. X-ray studies of the esophagus with contrast medium showed a markedly dilated esophagus and the presence of an intraluminal foreign body that measured 15 by 7 cm. (Fig. 4). Cardiospasm was suggested by the roentgenologist. At the time of esophagoscopic examination (H. A. A.), a large, intraluminal polyp was noted with an attachment arising from the posterior wall of the esophagus, about 10½ inches from the upper teeth. Fairly large blood vessels were noted on the surface of the tumor stalk. On April 2, 1955, a transpleural excision of the lesion was accomplished through a right fifth interspace incision (F. H. E.). The tumor extended from its attachment just above the level of the azygos vein to just above the diaphragm. The esophagus was markedly dilated throughout this distance. The tumor measured 14 by 7

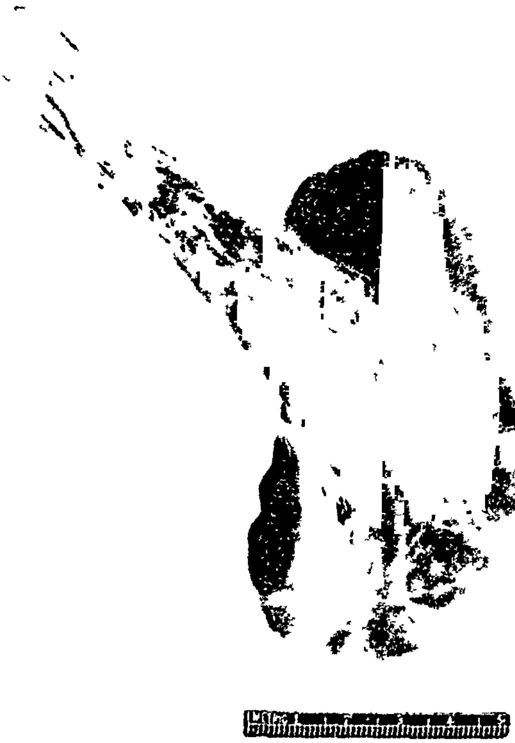

Fig 2—Lobulated fibrolipoma with long pedicle of attachment

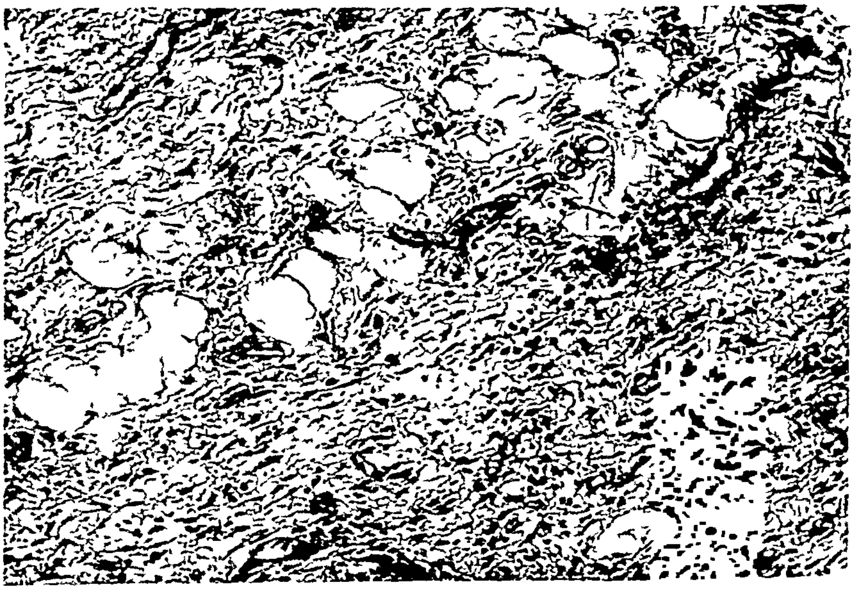

Fig. 3—Representative area of fibrolipoma (hematoxylin and eosin, ×155)

by 5 cm. The mucous membrane was intact over the somewhat lobulated surface with the exception of an area of ulceration at the distal end of the tumor (Fig. 5). The microscopic character of the tumor is shown in Fig. 6.

The patient's early postoperative course was complicated by enteritis due to *Micrococcus pyogenes*, which responded quickly to erythromycin therapy. She had no difficulty swallowing. X-ray studies of the esophagus 2 weeks after operation showed the esophageal dilation to persist, with some delay of barium at the esophagogastric junction. Studies of esophageal motility showed that the gastroesophageal junctional region failed to participate in the swallowing reflex and also showed failure of the primary peristaltic wave. Methacholine chloride (Mecholyl) injected subcutaneously did not elevate the intraluminal pressure of the esophagus. It was impossible to conclude definitely whether this patient had primary achalasia also, or whether these findings were all due to prolonged esophageal dilation and operative trauma.

For 5 months the patient ate normally and during this period she gained 20 pounds. In October, 1955, she again noted the onset of dysphagia and regurgitation similar to her preoperative symptoms. She was re-examined and roentgenograms of the esophagus showed evidence of some dilation but very little delay of barium at the esophagogastric junction. Repeat studies of esophageal motility showed the cardia to be nonfunctioning and incompetent, and, in comparison to the previous studies, suggested a "muscular or nervous" degeneration of the esophagus with changes too extensive to be accounted for by the operation. Esophagoscopy revealed the lower portion of the esophagus to be atonic with redundancy of the esophageal mucosa. A sound, size 30 Fr., was passed into the stomach without difficulty.

In February, 1957, this patient reported her health to be good and she no longer suffered from dysphagia.

ETIOLOGIC ASPECTS

It has been proposed that in view of the usual high midline origin of these strange esophageal lesions they may represent a developmental anomaly of one of the branchial arches.[6] There is little support for such conjecture. Practically speaking, with some proliferation of tissue in the loose submucosal area of the proximal esophagus, one can visualize how the propulsive movements of swallowing could play a significant role in the development of these tumors. From the available gross and histologic evidence, it seems most reasonable to include these lesions as true neoplasms rather than unusual inflammatory reactions.

PATHOLOGIC FINDINGS

Pathologically, these tumors comprise a somewhat heterogeneous group. This is apparent from the variety of pathologic terms employed in the literature to classify these unusual lesions, including fibromas, lipomas, myxomas, leiomyomas, myomas and various combinations of these terms. Grossly, the lesions are all elongated to some degree, presumably because of the molding effect of the esophageal wall. The surface of the tumor may be irregular and lobulated, and occasionally it is distinctly a multilobed structure arising from a common pedicle. Multiple tumors have been found to occur in the same patient.[5] The mucous membrane of squamous epithelium is usually intact over the surface of the tumor. Ulceration of the surface may occur secondary to the trauma of swallowed food, or as a result of peptic digestion of the distal end of the growth which may protrude through the cardia. Microscopically

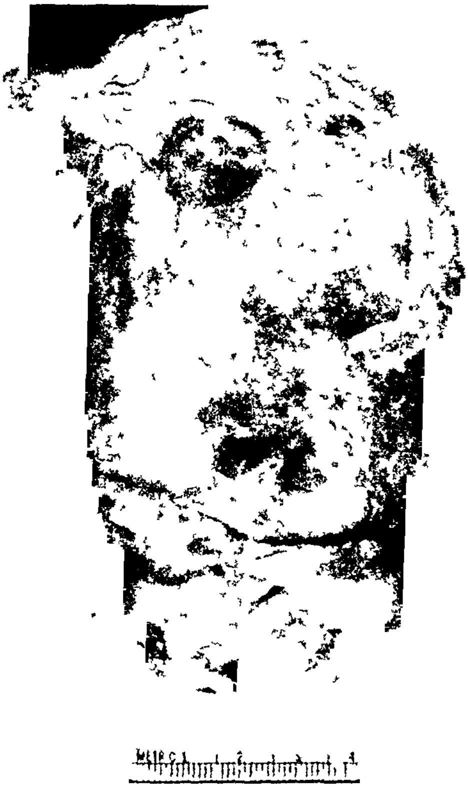

Fig. 5.—Intraluminal polyp with ulceration at its distal end.

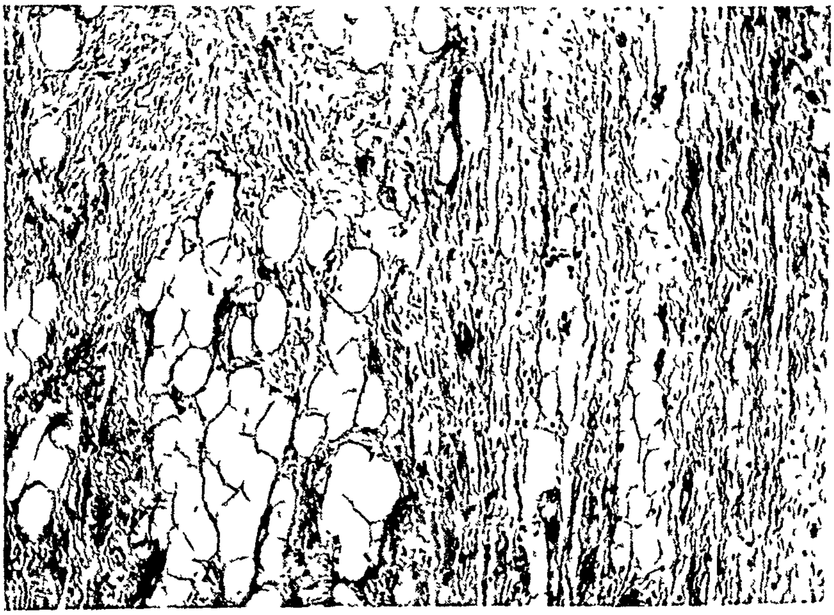

Fig. 6.—Intermixture of fatty tissue and fibrous stroma typical of benign intraluminal esophageal polyps (hematoxylin and eosin, ×100).

was used in one case in this series. However, vessels in the pedicle may present problems of hemostasis and it would seem more reasonable to approach this usual site of attachment of the pedicle more directly through a cervical incision Occasionally the attachment will be in the thoracic portion of the esophagus and, depending on the level, an approach through either the right or left pleural space should permit easy removal of the intraluminal tumor.

PROGNOSIS

Without a diagnosis and removal many persons having these benign intraluminal tumors have died from complications owing to the lesion. Totten and associates noted that of 40 patients having such growths, a third died as a direct or indirect result of the tumor and in most cases the diagnosis was incorrect Complete respiratory obstruction at the laryngeal level may be produced by regurgitation of the mass Chronic esophageal obstruction with dilation of the lumen can be associated with intermittent aspiration of esophageal contents and pneumonitis, bronchiectasis pulmonary abscess or other serious suppurative complications

With surgical removal of the growth, one would expect complete relief of symptoms This has been true in a majority of patients but some have continued to have symptoms related to esophageal dysfunction The dilated esophagus may not assume normal proportions, as illustrated in the case of the most recent patient in this series This young woman continued to complain of intermittent dysphagia and regurgitation, and x-ray studies revealed persistent, moderate dilation of the esophagus without much delay in transit of the barium at the esophagogastric juncture Studies of esophageal motility revealed some characteristics of cardiospasm but these were not sufficiently typical to be diagnostic One could conjecture a secondary degeneration of myenteric plexuses from the prolonged esophageal dilation, or a primary achalasia as an associated condition Unfortunately a portion of the esophageal wall of this patient was not removed for study She continued to have dysphagia and occasional regurgitation, repeated studies of esophageal motility a few months later revealed even more abnormalities of function and suggested a "progressive muscular or nervous degeneration" It is possible that irreversible changes in the wall of the esophagus occurred with its prolonged dilation Beeler and associates[1] reported that cardiospasm is not uncommonly associated with long intraluminal tumors Studies of esophageal motility do not support such a coincidence and we would suspect these patients to have problems similar to those of the patient described in the second case reported herein

SUMMARY

Benign intraluminal esophageal tumors may result in distressing symptoms for the patient and can present perplexing diagnostic problems for the physician X-ray and esophagoscopic examinations may precisely delineate the origin and extent of the lesion and surgical removal can be accomplished with these tumors present a mixture of loose, fibrous tissue associated with myxomatous and fatty changes. Inflammatory reaction may be evident in the vicinity of surface ulceration. It would seem most descriptive and convenient to designate the group histologically as fibrolipomas.

CLINICAL MANIFESTATIONS

The commonest clinical manifestations of these intraluminal, polypoid growths result from obstruction of the lumen of the esophagus. Dysphagia, regurgitation and vomiting may be intermittent, but the reduction in intake of food is such that significant loss of weight usually occurs. The average loss in this series of patients was 25 pounds. Most of the patients complained of substernal distress of some type. An uncomfortable feeling of substernal fullness is a common complaint, but some patients describe pain very suggestive of angina. Respiratory symptoms, consisting of cough, dyspnea, recurrent infection and even hemoptysis, may arise as a result of aspiration of esophageal contents or compression of the tracheobronchial tree. Ulceration of the surface of the tumor may manifest itself in melena or hematemesis. The most dramatic evidence of the lesion is its appearance in the patient's mouth with coughing or regurgitation.

DIAGNOSIS

As a group, these patients have presented perplexing diagnostic problems. The history may be only suggestive, or it may be diagnostic with the story of a regurgitated tumor on one or more occasions. This was true in 4 of the 6 patients in this group. A roentgenogram of the thorax may exhibit widening of the mediastinal shadow as a result of the tumor and associated dilation of the esophagus. Ingested barium may divide at the level of the mass and delineate its borders precisely. Along with this evidence the roentgenologist may then note movement of the tumor and irregularities of the peristaltic wave above the tumor at the level of attachment of the pedicle. Occasionally the esophagus may be greatly dilated and the intraluminal tumor can be mistaken for a large foreign body. This is illustrated by the fact that 4 of the 6 patients in this series were thought to have cardiospasm.

Esophagoscopy may be the most precise means of diagnosis. Some of our patients, as well as many of those whose cases are reported in the literature, had undergone this procedure on more than one occasion. The growth was overlooked because the examiner was misled by the intact mucous membrane over the intraluminal tumor. The endoscopist may first discover the tumor at its distal end. The remainder of the tumor, its pedicle and attachment may then be visualized by appropriate maneuvers.

TREATMENT

The indicated treatment is surgical removal. These tumors have been excised by oral, cervical, and thoracic approaches. The oral route may be technically feasible if the tumor can be delivered into the mouth; this approach

IVALON GRAFT TO BRIDGE CORONARY ARTERY OCCLUSION

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In one hundred consecutive autopsy specimens we found that the majority of patients who die of coronary heart disease have a short intraluminal obstruction near the origin of a major artery. The artery distal to the occlusion is frequently sufficiently free of compromise of lumen to supply adequately enough blood flow to nourish the myocardium. The ideal surgical attack on this problem would be to open the coronary artery at the point of obstruction, ream out the atherosclerotic block, and close the incision. However, currently, the small size of the vessel and the technical problems involved militate against this direct approach, enticing though it is. Further, if the obstruction is complete, death of peripheral myocardium would make such an endarterectomy of little or no use to the infarcted area. Also, atherosclerosis is not generally confined to a solitary coronary artery but is a generalized process and further obstruction can occur subsequently in another artery.

Many other methods of revascularization have been suggested. In our hands the most promising technique is cardiopneumopexy, developed in this laboratory, a desirably simple procedure with a low mortality rate equal to only 6 months of medical treatment. This method consists of wide incision of the pericardial sac, phenol epicardiolysis, light asbestos poudrage and suturing the lung to the myocardium or inner surface of the adjacent pericardium.[1, 2] Over the past 5 years we have studied this procedure extensively utilizing 187 dogs and have employed it clinically 21 times with encouraging results. However, it is no panacea, but a valuable adjunct to existing medical therapy for coronary heart disease. Furthermore, a minimum of another 10 years of clinical observation is necessary before final conclusions as to its efficacy can be drawn.

In an experimental effort to attack this problem of coronary occlusion in more direct fashion, we proposed an Ivalon graft as a shunt between the left subclavian artery and a coronary artery beyond the point of an acute obstruction.

TECHNIQUE

In this procedure the left chest wall is opened by posterolateral incision through the fourth interspace. The pericardium is opened widely and the

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This research was carried out under a grant of the Washington State Heart Association. The research laboratories of the Seattle Veterans Administration Hospital were used for the experiments.

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**Research Assistant.

minimal risk. With excision of the growth, complete relief of symptoms is usually attained, but derangement of esophageal function may prevent a completely satisfactory result for the patient.

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of anastomosis of graft to coronary artery This obstacle was present in each instance in spite of the use of heparin at the time of surgery The advisability of using more prolonged anticoagulant therapy, employing either heparin or Dicumarol, was considered but rejected

Actually a higher percentage of dogs survived this procedure than in our control series in which 3 out of 4 dogs succumbed to ligation of the left anterior coronary artery[2]

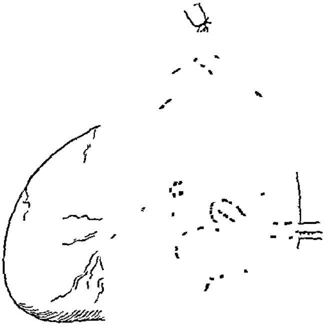

Fig 2 —This depicts the graft in situ The left anterior descending coronary artery is ligated securely proximal to the anastomosis to produce an acute coronary occlusion of substantial dimensions in a heart not given the advantage of gradually developing anastomotic channels

MATERIAL

Ivalon is a polyvinyl sponge found to be biologically inert and, when implanted in tissue, is rapidly invaded by a noninflammatory type of fibrous tissue Ivalon is formed by hardening foamed polyvinyl alcohol with formaldehyde

Preparation of the Ivalon graft has been standardized in this laboratory Ivalon surgical sponge is supplied commercially sealed in a plastic film bag with a small amount of glycerol (hygroscopic agent) and a trace of the formaldehyde that was used in polymerization Before use it must be thoroughly washed free of these contaminants This may be done by leaving in running tap water for several hours or by rinsing and wringing 30 to 50 times

After thoroughly washing it has been our practice to allow the sponge to air dry for several days, after which time it is quite rigid For this particular use we cut the sponge into slabs that measure some 3/16 inch thick when wet The slabs are scissor cut into strips the length of the sponge and 1/2 inch wide After soaking in warm water, these strips are ready for fabrication

To adapt the grafts to the wide variations in anatomy they are made into a tube that tapers from 1/8 inch to 7/64 inch in 2½ inches and then remains constant for 1½ inches The wall thickness is approximately 1/32 of an inch This configuration is obtained by wrapping the strips in a spiral around a polished brass form, compressing the Ivalon with a 0 006 inch strip of polyvinyl film twice, and fixing the Ivalon with heat (boiling water).

subclavian artery isolated in the neck. The left anterior descending coronary artery is elevated from its bed. The subclavian artery is severed between patent ductus clamps, the distal end doubly ligated, and the proximal end anastomosed to the large end of the tapered Ivalon graft. Then with a scalpel, a nick is made in the coronary artery. The smaller end of the graft is inserted distally and fixed in position by a simple silk ligature. The coronary artery is then tied off proximad the anastomosis (Fig. 2). The lung is expanded and the chest closed in the usual fashion.

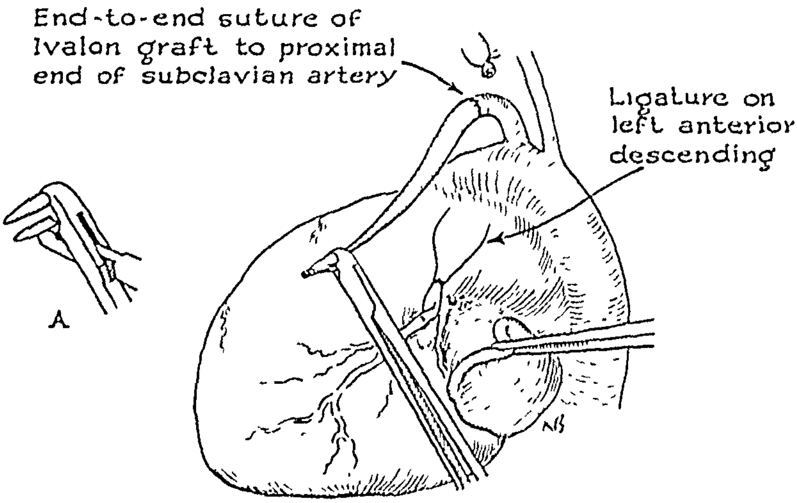
End-to-end suture of Ivalon graft to proximal end of subclavian artery
Ligature on left anterior descending
A
NB

Fig. 1.—This sketch denotes the view of the heart obtained through a left posterolateral incision through the fourth interspace. One can see the short left coronary artery with its two major branches, the left anterior descending and the circumflex arteries. These are readily visible when the auricle is elevated. In this particular technique the subclavian artery is cleanly separated from its surrounding tissue in the neck and severed between patent ductus clamps. The distal end is doubly tied and permitted to retract into the neck. The proximal end is anastomosed end-to-end to the large end of the Ivalon graft. The left anterior descending coronary artery is then elevated from its bed and cleaned over a distance of approximately 1.5 cm. and a transverse nick made in it on the anterior surface for introduction of the distal end of the graft.

A very useful instrument, developed by one of the authors (D. M.), has been found to be of considerable help in introducing the distal end of the Ivalon graft into the very small-bore coronary artery (Fig. 1). It has the advantage of holding the coronary artery wide open and permitting rapid introduction of the graft.

In this series we operated on 18 dogs. The first 5 dogs constituted efforts to evolve a technique and actually are not considered a part of the present statistics. Of the next 13 dogs, on whom the standardized procedure was used, 7 or approximately one half died the day of surgery, generally from ventricular fibrillation when the left anterior descending coronary artery was tied. The remainder were sacrificed 4 to 10 weeks later. In 4 of these there was evidence of at least subendocardial infarction and, in some instances, more extensive infarction of the myocardium. The Ivalon graft usually was patent and contained at the most some jelly-like material. In only one instance was there obstruction by thrombus of the subclavian artery-Ivalon graft anastomosis. It was the impression of the investigators that the problem in this technique is to get blood to flow through the peripheral myocardial field, beyond the point

PULMONARY ARTERIOVENOUS FISTULA ASSOCIATED WITH CAPILLARY TELANGIECTASIA (RENDU-OSLER-WEBER DISEASE)·

REPORT OF A CASE ILLUSTRATING USE OF METAL CASTING FOR DEMONSTRATING THE LESION

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PULMONARY arteriovenous fistulas are congenital hemangiomatous malformations of the pulmonary vascular bed. In recent years, a close relationship between capillary telangiectasia (Rendu-Osler-Weber disease) and pulmonary arteriovenous fistulas has been recognized. In a series of 16 cases of pulmonary arteriovenous fistulas studied at The New York Hospital, 6 patients (38 per cent) had characteristic mucosal and skin lesions of Rendu-Osler-Weber disease.[1, 2] In addition, 2 patients in the series, although without capillary hemangioma, gave a history of such lesions in members of the family.

In 1952, a method of preparing metallic casts of anatomic specimens was described[3] Essentially it consists of the pouring of a low melting point fusible metal into an anatomic specimen, allowing it to harden and then digesting the tissue with strong alkali. Pulmonary arteriovenous fistulas, being vascular malformations with afferent pulmonary arteries and efferent pulmonary veins, are admirably suited for such studies; particularly, because their gross anatomic features provide more information about the disease than does histologic study.[1] Since 1953, a total of seven excised pulmonary arteriovenous fistulas, three segmental and four lobar, were studied after casting. Six of the cases with casts of the operative specimen were previously reported[1, 2, 4]; the sixteenth patient with a pulmonary arteriovenous fistula and the seventh, with study of the specimen after casting, had disabling epistaxis due to nasal capillary telangiectases and is herein recorded.

CASE REPORT

A 46 year old butcher (N. Y. H. No. 762570) was admitted to the hospital on March 17, 1957, with chief complaint of nosebleeds. Ever since childhood the patient had had epistaxis. At first these occurred about once a month but in the past year they had

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We use one 10-inch length of 0.006 by ½ inch polyvinyl film for each graft and one 8½-inch strip of Ivalon, ³⁄₁₆ inch thick and ½ inch wide.

Some silicone-lined grafts were prepared but these seemed to be of no greater value than the standard graft.

SUMMARY

In an effort directly to attack the atherosclerotic intraluminal obstruction in coronary occlusion we have investigated the use of a tapered Ivalon graft, shunting blood from the left subclavian artery to the left anterior descending coronary artery distal to a complete acute obstruction. More dogs have lived following this procedure than in the control series of ligations. Nevertheless, in none of these dogs was circulation through the graft maintained for any great length of time. The problem of anastomosis of grafts to small-bore arteries has not been solved as yet.

At this stage we feel that an Ivalon graft to bridge such a block is not a clinically feasible solution to coronary artery obstruction and that cardiopneumopexy is a more successful and desirable procedure. Furthermore, coronary disease is multicentric and further occlusions could occur in another artery. However, we hope that eventually direct attack on the obstruction with removal of the atherosclerotic plaque will be possible to aid those patients with an incomplete block and in whom the myocardium supplied by that vessel is intact as yet. This series is intended as a steppingstone toward that goal.

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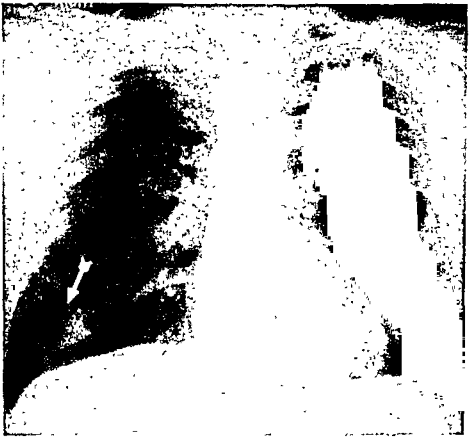

Fig. 2A.—Conventional frontal teleoroentgenogram of the chest showing a globular mass (arrow) with hilar vascular connection in the outer portion of the right lung.

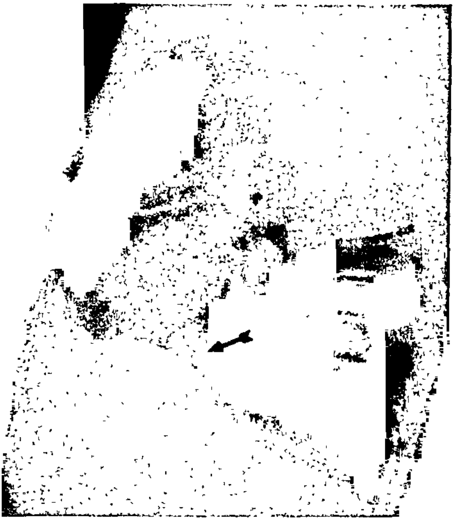

Fig. 2B.—Lateral roentgenogram locates the lesion to be in the anterior segment of the right lower lobe (arrow).

become more frequent, usually being associated with the loss of "about a cup" of blood. Packing and cautery of the nose had abated the bleeding at first but, recently, these measures had failed.

Seventeen years prior to admission, the patient had had a routine roentgenogram of the chest after enlistment in the National Guard, and when a "spot" was found in the right lung, the military service was terminated. Since then frequent x-ray studies of the chest had been made and the lesion had remained unchanged. At one time exploratory thoracotomy had been recommended but had been declined by the patient because symptoms referable to the respiratory system were absent. The patient's father had died at the age of 86 years, cause unknown. His mother, age 74 years, was still alive and she, 5 siblings, and several cousins had had epistaxis.

On physical examination the patient was pale, well developed and nourished. There was no cyanosis or clubbing of the fingers. Scattered small clusters of cherry red angiomata were seen over the face and trunk. The nose contained crusted blood and capillary angiomata; a few (less than 1 mm.) capillary angiomata were present over the nose and cheeks. The tongue (Fig. 1) also contained many angiomata. The blood pressure was 150/80 mm. Hg; the heart was regular and there were no murmurs. A loud systolic bruit was heard at the lower end of the right anterior chest; otherwise, the examination was normal.

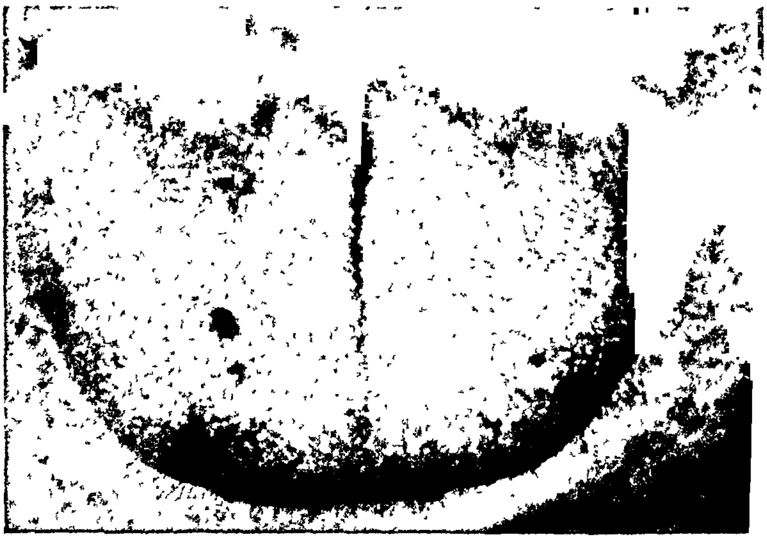

Fig. 1.—Photograph showing scattered capillary hemangiomata of the tongue characteristic of Rendu-Osler-Weber disease.

Laboratory data revealed secondary anemia with hemoglobin 9.8 Gm. per 100 c.c.; hematocrit, 29 per cent; bleeding and clotting times were normal. A roentgenogram of the chest (Fig. 2) showed a normal-sized heart. An oval 4.5 by 2.5 cm. mass with intimate relations to the vascular structures of the right hilus was seen at the right base. The lateral view (Fig. 2B) localized the lesion in the anterior segment of the right lower lobe. Angiocardiography (Fig. 3) clearly established the diagnosis of pulmonary arteriovenous fistula involving the anterior basilar segment of the right lower lobe. Treatment of the hemangiomata of the nasal mucous membrane by cauterization was followed by cessation of epistaxis. After transfusion, on March 22, 1957, segmental resection of the anterior segment of the right lower lobe containing the arteriovenous fistula was performed. A low melting point alloy cast of the specimen was made (Fig. 4). The patient was improved, and was discharged on March 31, 1957. Since then, recurrence of epistaxis, despite treatment with testosterone, packing, and x-radiation to the nasal mucosa, resulted in readmissions on April 9 and May 13, 1957.

COMMENT

Excision of a pulmonary arteriovenous fistula, once the diagnosis is established, is advisable even in the absence of respiratory complaints This is because of the high incidence of brain abscess, cerebral thrombosis, and fatal hemoptysis in such patients. The risk from complications is far greater than the mortality and morbidity of lobectomy or segmental lung resection.[1, 2]

A glance at the cast of the pulmonary arteriovenous fistula made from the excised segment of the right lower lobe readily reveals the complexity of such lesions (see Fig. 4) Pulmonary arteriovenous fistulas are not merely communications between an afferent end pulmonary artery and vein. Rather, they are intricate lesions with centrally dilated paper-thin globular connections that frequently involve the entire blood supply of the lobe or segment. In addition, arterial and venous communications from neighboring chest wall, bronchus, or lung may occur. Indeed, the rich admixture of oxygenated blood from the bronchial arteries or pulmonary veins may prevent development of cyanosis.[1, 2]

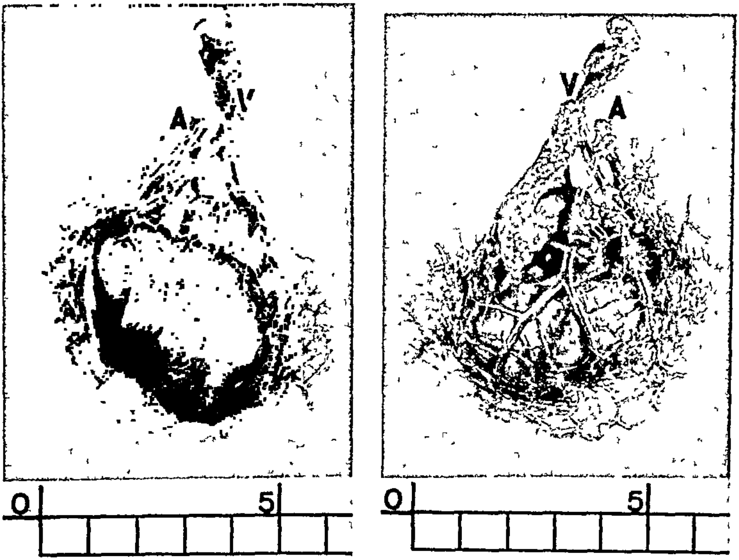

Fig 4.—Alloy cast of pulmonary arteriovenous fistula. *Left*, Anteroposterior view. *Right*, Posteroanterior view *A* is the pulmonary artery, *V*, the vein

Normally, the intact pulmonary capillary bed acts as a sieve and noxious agents, especially bacterial, are prevented from reaching the systemic blood supply In the presence of right-to-left shunting of blood that occurs with

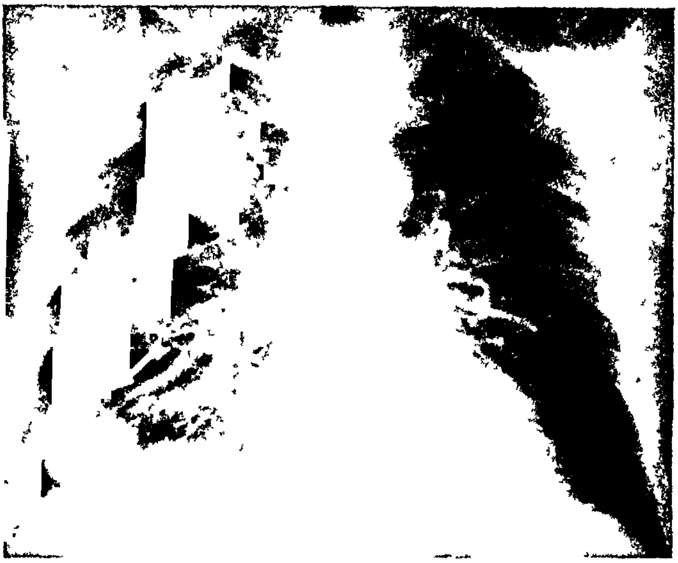

Fig. 3A.—Frontal teleoangiocardiogram, made 2½ seconds after the beginning of injection, reveals the afferent pulmonary arterial blood supply to the fistula (arrow).

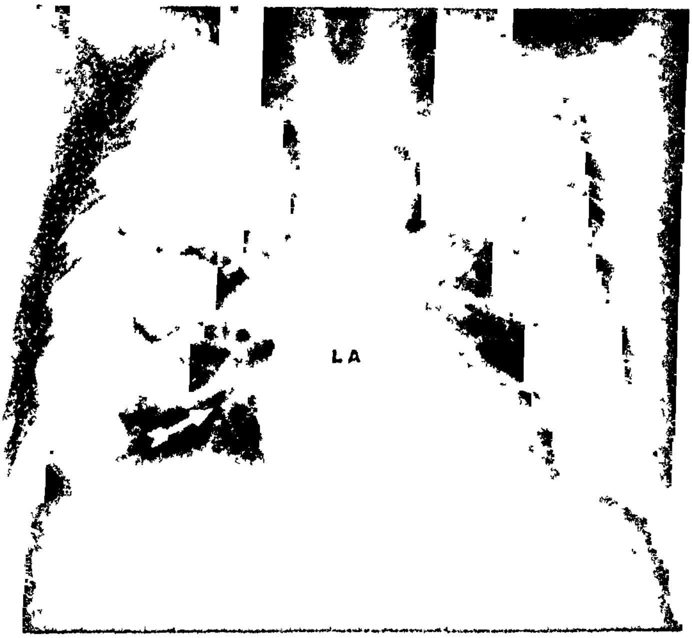

Fig. 3B.—At 5½ seconds, segmental pulmonary veins can be seen draining into the left atrium (*LA*).

SOME PROBLEMS IN EXTRAPLEURAL PNEUMONECTOMY FOR TUBERCULOUS EMPYEMA AND DESTROYED LUNG

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Oteen, N.C.

THE destroyed lung, due to tuberculosis complicated by empyema, has been an age old problem. Estlander, in 1879, and Schede, in 1890 attacked the parietal portion of the empyema while Fowler and Delorme contributed to the visceral portion of empyema surgery. Experience gained during World War II marked another step in the surgical treatment of empyema, that of visceral decortication and early expansion of the underlying lung. More recently the combined procedure of excision of the empyema sac and the underlying diseased lung through the extrapleural plane has been the method of attack in these problems.[1] Contralateral pulmonary disease, decreased pulmonary function, resistance of the tubercle bacilli to antituberculous drugs, and the immeasurable immunologic status all contribute in the response of the patient to any given regimen of treatment. Those patients coming to extrapleural pneumonectomy and excision of the empyema "in toto" are a chosen few. On the other hand, many patients have been denied such definitive therapy because of complications arising during prolonged preparation or because of refusal to accept surgery.

A study of those patients undergoing pleuropneumonectomy as definitive therapy was undertaken. This paper is directed (1) to evaluate whether the extensive operative procedure of pleuropneumonectomy in complicated pulmonary tuberculosis yielded a sufficient number of rehabilitated patients, and (2) to examine the causes of complications in an endeavor to reduce the operative and late morbidity and mortality. Decortication and subtotal pulmonary resection will be discussed in another paper.

Both far advanced pulmonary tuberculosis and tuberculous empyema carry a high mortality rate. This mortality rate is further increased by the presence of bronchopleural fistula and mixed empyema. Alling and co-workers, in a study of far advanced pulmonary tuberculosis in the prechemotherapy era, arrived at a figure of 70 per cent mortality in a 10 year follow up, with 20 per cent mortality in the first year. Empyema without extensive pulmonary disease also carries a high mortality rate of 67.6 per cent within 5 years after the onset of empyema and a recovery rate of only 19 per cent.[3] Klassen and associates[4] reported a mortality rate of 38 per cent in empyema treated by thoracoplasty

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pulmonary arteriovenous fistulas, septic emboli may readily reach organs like the brain. A recent report of a liver abscess in a patient with a pulmonary arteriovenous fistula indicates that other organs may also be the sites of abscesses.[4]

SUMMARY AND CONCLUSION

Angiocardiography established the diagnosis of a pulmonary arteriovenous fistula of the right lower lobe in a 46-year-old man with epistaxis due to capillary telangiectases (Rendu-Osler-Weber disease). Segmental excision of the fistula and study of the specimen by making a metallic cast disclosed that fistulas are complex and bizarre hemangiomatous malformations.

Excision of pulmonary arteriovenous fistulas is recommended because, when they are removed, isolation of noxious agents in the pulmonary capillary bed prevents cerebral abscesses and thrombosis. The right-to-left shunting of blood is also corrected. Finally, since the fragile paper-thin fistulas have a tendency to rupture, removal will prevent fatal hemoptysis.

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Comment: This case illustrates the points that preliminary closed thoracotomy drainage does not necessarily lead to infection of the operative hemithorax and that protection against contralateral spread can be obtained by closed thoracotomy. A short course of preoperative drug therapy in stable disease was sufficient.

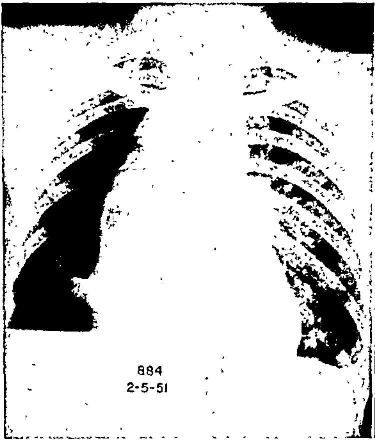


Fig. 1.

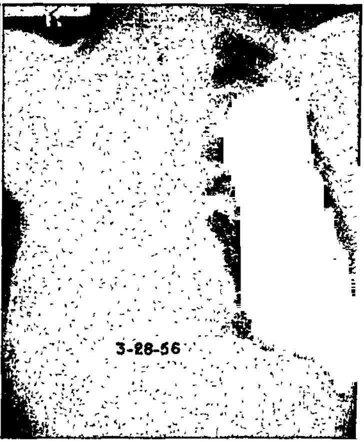


Fig. 2.

Fig. 1.—Case 1. Admission film shows empyema, bronchopleural fistula, and cavitary pulmonary disease. There is scattered disease on the left side.

Fig. 2.—Case 1. Postoperative film showing clear left lung field and right pneumonectomy 5 years postoperative.

Case 2.—C. S., a 29-year-old white man, was admitted to the hospital in 1949 with pulmonary tuberculosis and right pneumothorax. After 4 months of drug therapy he left the hospital against medical advice. He was readmitted in 1950 with tuberculous empyema and bronchopleural fistula (Fig. 3). After one week of streptomycin therapy an extrapleural pneumonectomy was performed on June 29, 1950. Gross pleural contamination of the pleural space occurred during the operative procedure. Postoperative bronchopleural fistula and empyema ensued and a three-stage space-obliterating thoracoplasty was performed. The patient was discharged 28 months after surgery. Follow-up roentgenogram demonstrates stable disease in the remaining left lung (Fig. 4). The patient is now working.

Comment: This case illustrates the following classical points. Spontaneous pneumothorax in a tuberculous patient may lead to empyema and bronchopleural fistula. Inadequate preoperative drug therapy, operation on unstable disease, and operative pleural contamination can lead to major postoperative complications. Established long-term drug regimens and adequate preoperative stabilization of pulmonary disease can prevent many of the complications. Postpneumonectomy empyema and bronchopleural fistula necessitate major collapse therapy and prolonged hospital care.

Connolly and Forsee[5] reported a mortality of 18 per cent in pulmonary tuberculosis and empyema treated with thoracoplasty and streptomycin. These figures illustrate the gravity of active pulmonary disease complicated by empyema.

MATERIAL

From 1948 to December, 1955, 44 patients had extrapleural pneumonectomy performed for destroyed lung. Of these, 30 had chronic mixed empyema and bronchopleural fistula. Cases of acute empyema present an entirely different problem and are therefore excluded from this study. Fourteen patients had pleural symphysis from prior pleuritis or effusions, and a complete extrapleural procedure was necessary for the pneumonectomy. These cases are not included in the study. All except one patient had demonstrable contralateral disease.

Eighteen patients were prepared for pleuropneumonectomy by lesser surgical procedures such as closed thoracotomy, open thoracotomy or both, and 6 patients had thoracoplasty and 2 had further stages of Schede thoracoplasty. Since these procedures failed to obliterate the chronic empyema space and control the pulmonary disease, extrapleural pneumonectomy had to be performed. The mean length of preparation was 3 years.

COMPLICATIONS

The complication rate of pleuropneumonectomy, in comparison with other types of lesser pulmonary surgery in this series, was high. As a rule, patients undergoing this type of surgery do not represent the best surgical candidates.

The most serious complications were empyema, bronchopleural fistula, tuberculous spread to contralateral lung, distant spread of tuberculosis, and wound infection. Postoperative empyema developed in 17 of the 30 patients and 9 of these were demonstrated to have bronchopleural fistula. Contamination of the operative field by opening into the empyema was a definite factor in the development of postoperative empyema. Of 15 patients having operative contamination, 10 developed postoperative empyema. Seven patients developed spread to the contralateral lung or to distant areas and all of these cases were associated with the development of postoperative bronchopleural fistula and empyema.

Of the 17 patients who had postoperative empyema, 7 demonstrated drug resistance. Only 2 patients who had drug resistance did not have postoperative complications.

The following case reports illustrate some of the problems of the surgical procedure.

CASE 1.—F. F., a 29-year-old, acutely ill, Negro man, was admitted, in 1951, with extensive pulmonary tuberculosis of the right lung complicated with empyema and bronchopleural fistula. Minimal contralateral disease was present (Fig. 1). He was treated with closed thoracotomy drainage. After 50 days of streptomycin and PAS therapy, a right extrapleural pneumonectomy was performed on March 20, 1951. The postoperative course was uneventful and he was discharged from the hospital with a diagnosis of inactive tuberculosis. Follow-up roentgenogram (Fig. 2) demonstrates the status of the remaining lung.

CASE 3.—G. B., a 38 year old Negro man, with left fibrocavernous tuberculosis and bronchiectasis, complicated with empyema and bronchopleural fistula (Fig 5), was initially treated with closed thoracotomy and later by open thoracotomy in 1948. Minimal contralateral disease was present at the time of admission. Subsequently, three stages of thoracoplasty and a Schede thoracoplasty failed to arrest the disease. Streptomycin, Promin and Tb I were administered from 1948 to 1950. On Aug 2, 1952, under Aldinamide and PAS therapy, an extrapleural pneumonectomy was performed. The postoperative course was uneventful and he was discharged from the hospital in 1953. His present condition is excellent (Fig 6). The disease is inactive and he is working.

Comment. In the face of contralateral disease, stabilization of the lesser lesion was obtained by drug therapy and bed rest. Reduction of the empyema space was obtained by thoracoplasty to prevent contralateral spread through the bronchopleural fistula. This was the accepted method of treatment prior to the development of antituberculous drugs.[6] The residual active pulmonary disease on a small empyema sac were finally successfully subjected to extrapleural pneumonectomy. This is an exceptional instance where an open thoracotomy wound did not result in an infection of the postoperative thoracic space.

CASE 4.—G. H., a 40 year old Negro man, had had pulmonary tuberculosis since 1946 and had received right therapeutic pneumothorax for cavitary tuberculosis, resulting in chronic purulent empyema (Fig 7). He was admitted to the hospital on July 23, 1948. After 6 weeks of bed rest, he was given 10 days of daily streptomycin and pleuropneumonectomy was performed on Sept 20, 1948. Gross pleural contamination and an inadvertent opening of the pericardial sac occurred during the procedure. The postoperative course was complicated with pericarditis, empyema, bronchopleural fistula, and wound disruption (Fig 8). The patient died 2 weeks postoperatively. Histologically, the resected lung showed tuberculous pneumonia.

Comment. The preoperative drug coverage in this patient was inadequate according to our present standards. An acutely ill patient should have closed thoracotomy drainage of the empyema. The exudative phase of the pulmonary lesion should be controlled before surgery. This reduces the number and attenuates the tubercle bacilli, permitting surgery in a less acutely inflamed field. With the newer multiple drug choices, in contrast to the available drugs of 1948, acute tuberculous postoperative complications have been prevented in the majority of instances.

CASE 5.—T. M., a 33 year old white man, developed bronchopleural fistula and empyema following pneumonolysis in 1946. An open thoracotomy and two stages of posterolateral thoracoplasty were performed but the empyema and active parenchymal disease persisted (Fig 9). In view of the known bacterial resistance, an extrapleural pneumonectomy was performed on Dec 21, 1950, with no drug therapy. Postoperative contralateral spread developed and, 3 months later, a bronchopleural fistula was finally demonstrated. The patient died in irreversible pulmonary insufficiency and cor pulmonale 4 months after surgery (Fig 10).

Comment. The undiagnosed postoperative bronchopleural fistula was a factor in contralateral spread and final demise of the patient. Bronchopleural fistula is the most common cause of postoperative spread of tuberculosis. Early diagnosis and empyema drainage may have obviated this mortality. Open

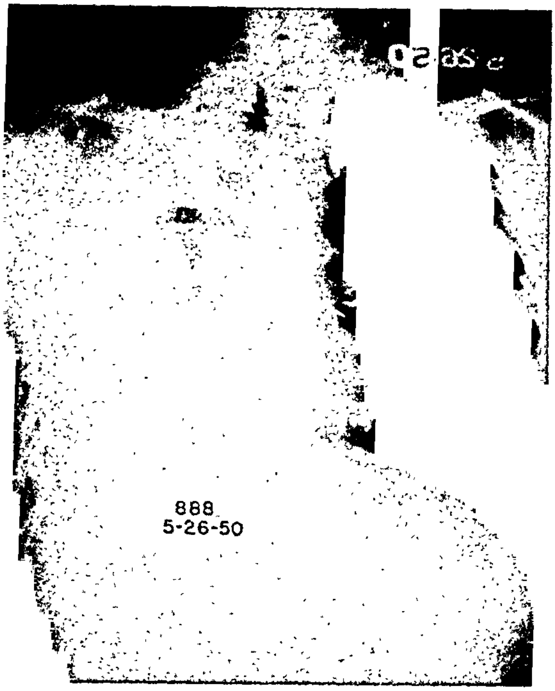


Fig. 3.

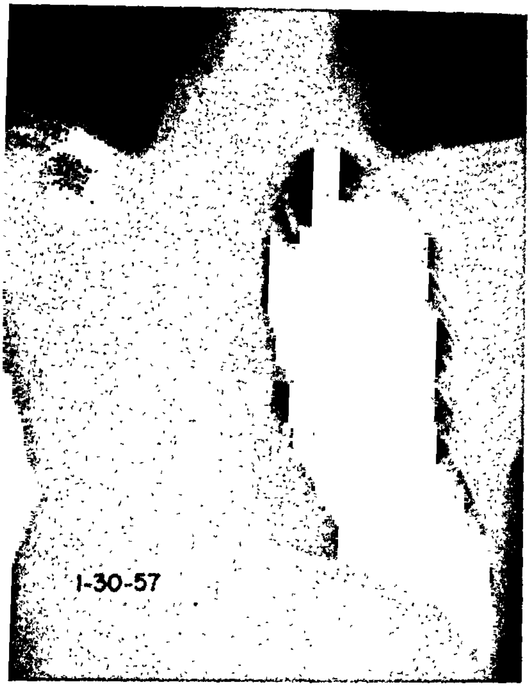


Fig. 4.

Fig. 3.—Case 2. Preoperative film shows right empyema, bronchiectasis and contracted lung.

Fig. 4.—Case 2. Follow-up film about 7 years after pleuropneumonectomy and thoracoplasty. Nodular but stable disease is seen.

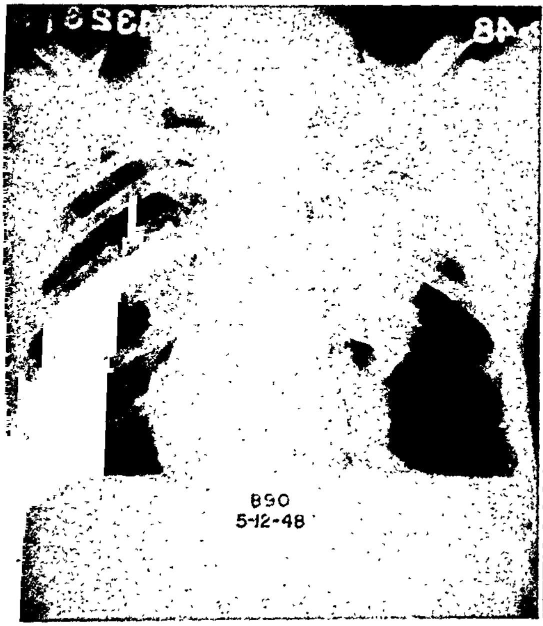


Fig. 5.

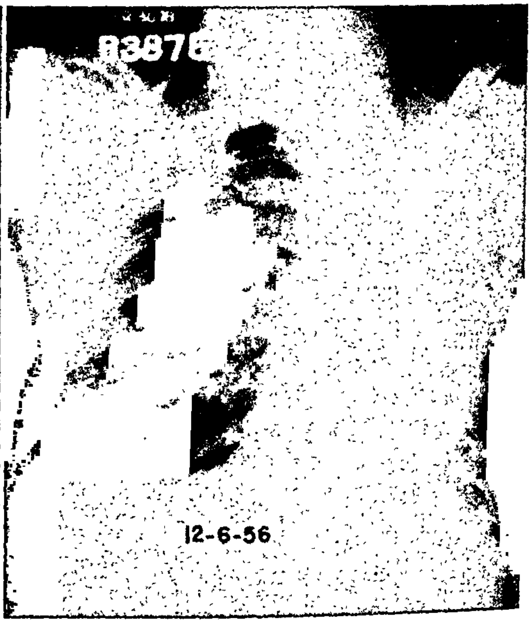


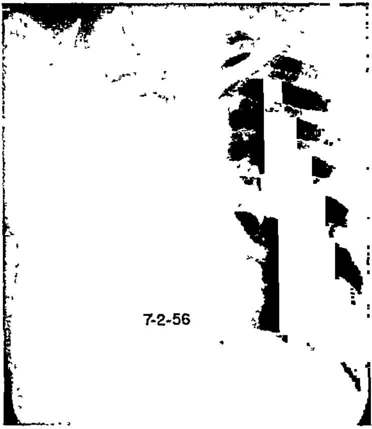
Fig. 6.

Fig. 5.—Case 3. Admission film shows destroyed left lung, empyema, and bronchopleural fistula. Right lung contains moderately active disease.

Fig. 6.—Case 3. Follow-up film shows inactive disease 4 years after pleuropneumonectomy and thoracoplasty.

thoracotomy preceding extrapleural pneumonectomy is almost always followed by infection of the thoracic space and therefore means of drainage should be established when pleuropneumonectomy is performed.

CASE 6.—E. W., a 36-year-old Negro man, was admitted to a state tuberculosis institution in 1954 with tuberculous empyema. He received streptomycin and isoniazid and had multiple chest aspirations. In March of 1956, he was transferred to VAH, Oteen, North Carolina. In July, a right decortication was performed. The operation was associated with excessive oozing from the raw surfaces, requiring 14,500 c.c. of blood before the operation was terminated. Postoperative bleeding continued and at re-exploration the chest was filled with clots; however, no bleeding points could be found. The lung was nonexpansible and there was bleeding through the endotracheal tube. An emergency pneumonectomy was performed. The patient continued to bleed postoperatively, and evidence of hypoxia was apparent. Blood transfusions and intermittent positive pressure breathing through an endotracheal tube did not relieve the situation. Fibrinogen determination, fibrinolysin level, blood volume, and prothrombin time were normal. The platelet count had dropped to 85,000 per cubic millimeter. The patient died 24 hours after the second thoracotomy.



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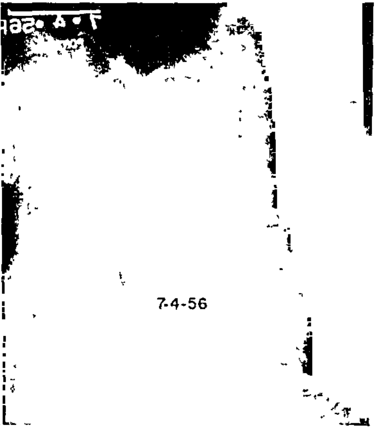

Fig. 12.

Fig. 11.—Case 6. Preoperative film shows right empyema and clear left lung.

Fig. 12.—Case 6. Postoperative film showing blood filled hemithorax with No. 34 Fr. catheters in place which were not adequate to evacuate the blood.

Comment: Postoperative hemorrhage in major thoracic procedures is not an uncommon complication. Bleeding may be from open vessels which can be mechanically controlled; however, in oozing problems of various known and unknown causes, therapy becomes more difficult. Citrate excess, calcium deficiency, prothrombin deficiency or deficient prothrombin regeneration, fibrinogen deficiency or increased fibrinolysin titer, platelet deficiency, hypoxia, etc., as probable causes are all under study in various laboratories. Perhaps, in the near future, simple laboratory methods to determine the defects of clotting or bleeding and substitution therapy may become available to the control of unexplained hemorrhage during and after major thor ons.

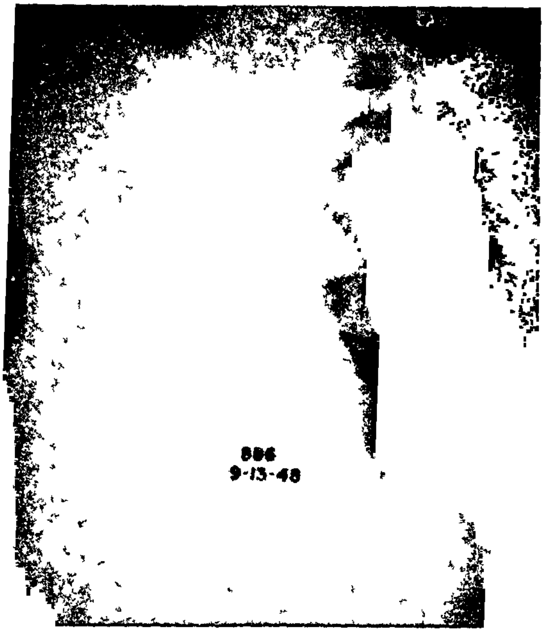

Fig. 7

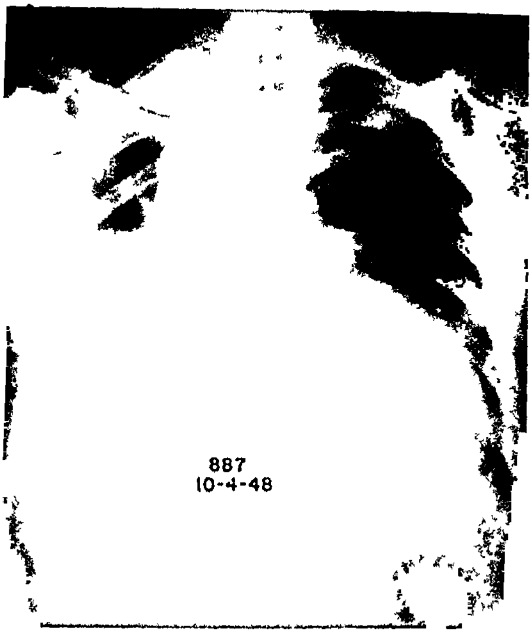

Fig 8

Fig 7.—Case 4 Preoperative film showing right empyema. The pneumonic lesion under the empyema was not detected from x-ray examination Examination of the resected specimen showed pneumonic disease.

Fig 8.—Case 4. Postoperative film showing massive pericardial effusion, parascapular wound, and air-fluid level in the right chest

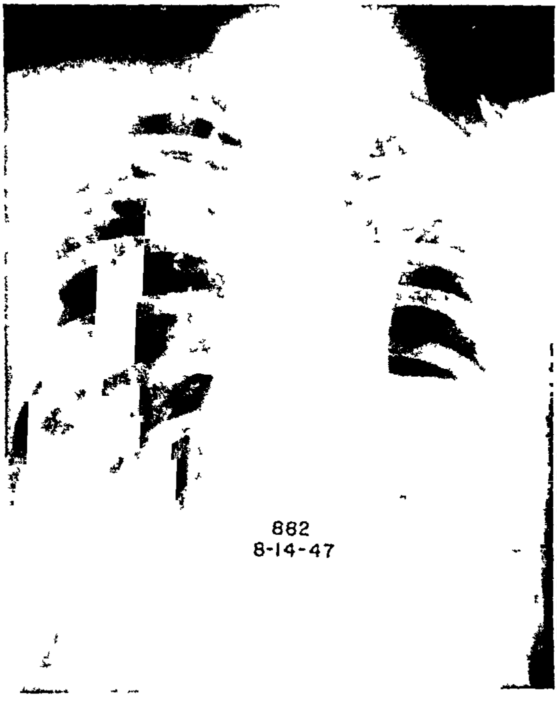

Fig 9

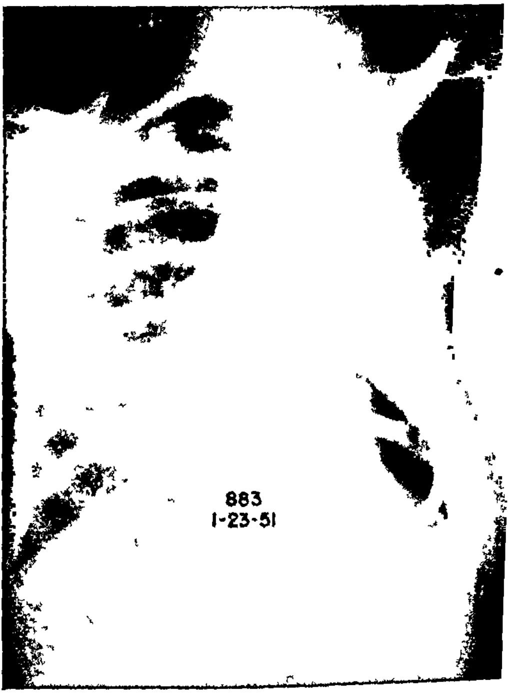

Fig 10

Fig. 9.—Case 5. Admission film shows left empyema and bronchopleural fistula and scattered lesions of the right lung.

Fig. 10.—Case 5. Postoperative film shows spread to right lung and empty left hemithorax due to bronchopleural fistula following pleuropneumonectomy.

operative procedures. Some had prior thoracoplasty. Pleuropneumonectomy, *although* a procedure accompanied by high complication rate, is a useful procedure for patients having no other recourse.

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RESULTS

A follow-up study of 30 patients with chronic purulent tuberculous empyema treated with pneumonectomy and total pleurectomy was carried out. The follow-up period extended from 9 years to 14 months. Nineteen, or 63 per cent, of the patients have attained a diagnosis of inactive tuberculosis and are rehabilitated in proportion to the motivation of the patient. Three patients are cripples and still have active disease. Eight patients, or 27 per cent, have died from the following causes: meningitis, pericarditis, postoperative sepsis, contralateral spread resulting in pulmonary insufficiency and postoperative hemorrhage.

SUMMARY AND CONCLUSIONS

1. Thirty patients who underwent extrapleural pneumonectomy for chronic purulent empyema and destroyed lung were analyzed as to complications and final results. Representative cases are presented.

2. Adequate antituberculous drug coverage and stabilization of the pulmonary disease are necessary prerequisites for this type of surgery.

3. Contamination of the operative hemithorax by opening into the empyema frequently leads to postoperative empyema and bronchopleural fistula. These complications can be reduced by adequate drug coverage.

4. Preparation of the acutely ill patient with empyema with the use of preliminary closed thoracotomy drainage has enabled proper stabilization of the disease. Pneumonectomy and excision of the empyema sac can be performed subsequent to this without incident. Open thoracotomy should be performed only when repeated and accurate closed intercostal drainage procedures have been inadequate to control the progress of pulmonary tuberculosis and empyema. If open thoracotomy is performed, the pleuropneumonectomy must be accompanied by a drainage procedure.

5. If the empyema has been chronic and the pulmonary disease stable for several months, a long period of drug therapy is not necessary.

6. In the presence of bronchopleural fistula with empyema, operative intervention should be done early in the course before the development of contralateral spread and drug resistance. If the general condition of the patient is unsatisfactory for major resection, complications can be prevented by space-obliterating thoracoplasty. Pleuropneumonectomy can then be performed in a less infectious field at a later date.

7. Postpneumonectomy bronchopleural fistula is the most common cause of postoperative contralateral pulmonary spread. The patient with postoperative spread should have careful examinations for determining the presence of bronchopleural fistula.

8. Two thirds, or 63 per cent, of the patients having chronic purulent tuberculous empyema and destroyed lung have obtained an inactive diagnosis and have been rehabilitated following extrapleural pneumonectomy. These patients were judged by the surgical staff as not being curable by other lesser

at a sensitivity of 100 or 200 mm. Hg per 4 cm. paper. Records were checked for electrical drift before and after each run. Pressure transducers were calibrated with a mercury manometer before and after each experiment.

Left ventricular pressures of the open chest dog were measured before, during, and after obstruction to inflow. Inflow obstruction was produced by inflation of the bag of a Foley catheter inserted through the left auricular appendage. The tip of the catheter lay in the left ventricle, and the bag itself lay inside the mitral ring. The bag was inflated with 40 to 55 c.c. saline to produce inflow blockage. It was planned to record pressures before, during, and following obstruction on three occasions for each animal. In 9 animals, left ventricular pressures during obstruction were measured also during infusion of l-norepinephrine in an amount sufficient to increase left ventricular systolic pressure by approximately 50 to 100 per cent. Right ventricular pressures were measured before, during, and after obstruction to filling produced by occlusion of the superior vena cava, inferior vena cava, and coronary sinus. In some animals, left ventricular pressure was measured during rapid exsanguination through a rubber tube inserted through the wall of the right ventricle. Pressures recorded during obstruction to inflow or bleeding represent an average of several heartbeats after reaching a maximal negative pressure at a fairly steady level.

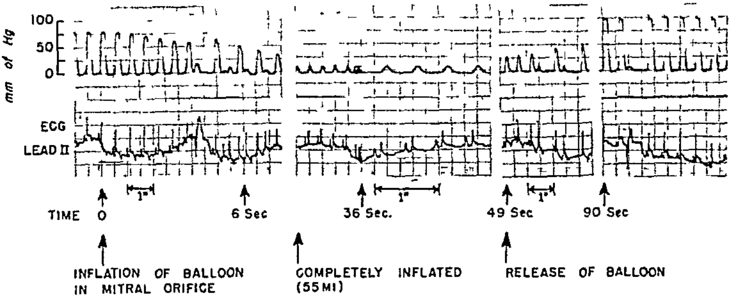

Fig 1.—Record of left ventricular pressure during obstruction to inflow by inflation of left atrial balloon. Pressure is 80/0 mm. Hg before obstruction and 8/–5 mm. Hg during maximal effect. Pressure is 95/0 mm. Hg after deflation of balloon. Paper speed is accelerated from 10 to 25 mm./sec. during maximum effect.

RESULTS

Results are given in Tables I to III and in Figs. 1 to 3. Satisfactory records of left ventricular pressure during obstruction to inflow (Table I) were obtained on 32 occasions in 13 animals. Adequate records of left ventricular pressure during l-norepinephrine infusion and inflow obstruction were obtained in 18 runs on 9 animals. The results are given in chronological sequence for each animal. It may be seen that control dia [illegible] wer

EFFECT OF INFLOW OBSTRUCTION AND RAPID BLEEDING ON VENTRICULAR DIASTOLIC PRESSURE

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BLOOM and Ferris,[1, 2] studying the excised beating rat heart immersed in liquid, demonstrated the development of negative left ventricular pressure during diastolic ventricular filling by suction. These authors[1] also found negative diastolic pressure in the right and left ventricles of the dog when inflow to the ventricle was obstructed. Brecher[3] demonstrated that the left ventricle of the dog was capable of exerting a sucking effect when its inflow was blocked. Kraner and Ogden[4] showed that the ventricle of the turtle was capable of exerting diastolic suction. The purposes of the present study were as follows: (1) to confirm and extend the above observations on the frequency and degree of negative ventricular diastolic pressure developed when there is obstruction to inflow; (2) to ascertain whether the magnitude of negative ventricular diastolic pressure developed during obstruction to inflow is influenced by infusion of l-norepinephrine; and (3) to determine whether negative ventricular diastolic pressure develops during diminution of ventricular filling as a result of acute hemorrhagic hypovolemia.

PROCEDURE

The studies to be described were performed upon 20 mongrel dogs, anesthetized with pentobarbital sodium, 30 mg. per kilogram of body weight. Studies were made with the animal on its back, the chest open, and with positive pressure respiration. Right and left ventricular pressures were measured through a No. 12 cannula with side perforations near the tip. The tricuspid valve was used as a zero reference level. Pressures were detected by means of a Statham P23Da strain gauge and were recorded upon a Sanborn Poly-Viso Electrocardiograph or upon an Electronics for Medicine Research Recorder. Diastolic pressures were recorded at the nadir of diastole. Paper speeds were 5 to 25 mm. per second. Right ventricular pressures were recorded at a sensitivity of 40 mm. Hg per 4 cm. paper; left ventricular pressures were recorded

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pressures were –2.5 to –20 mm. Hg. In only 5 of 9 animals were the peak negative pressures during norepinephrine infusion greater than those without norepinephrine infusion. In 3 of these 5 animals, maximum negative pressures reached during inflow blockage were not consistently greater during l-norepinephrine infusion. In 3 runs on 2 animals, systolic pressures also became negative during obstruction to inflow. An example of the effect of obstruction to left ventricular inflow upon left ventricular pressure is given in Fig. 1.

Table II gives the effect of obstruction to right ventricular inflow upon right ventricular pressure in 11 runs on 5 animals. Before inflow occlusion, right ventricular diastolic pressures were zero to 1 mm. Hg; during obstruction to right ventricular inflow, negative diastolic pressure was observed in each instance, the pressures ranging from –1 to –2.6 mm. Hg. Following the release of obstruction to inflow, right ventricular diastolic pressure became positive in each instance, ranging from 0.8 to 2 mm. Hg. An example of the effect of inflow occlusion upon right ventricular pressure is shown in Fig. 2.

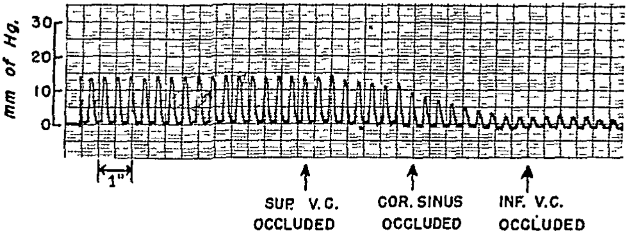

Fig 2.—Record of right ventricular pressure during obstruction to inflow produced by occlusion of coronary sinus, and superior and inferior venae cavae. Pressure is 14/1 mm. Hg before obstruction, and 2/–1 mm. Hg during obstruction

TABLE II. RIGHT VENTRICULAR PRESSURES, MM. HG, DURING OBSTRUCTION OF INFLOW

DOG	BEFORE OBSTRUCTION	DURING OBSTRUCTION	FOLLOWING OBSTRUCTION
5 6-57	17/0 8	1.2/–1	24/1
	20/1	2/–1	22/2
	22/1	1/–1	24/1.5
5-8 57	32/0	0/–1.8	26/1
No. 1	25/0.6	2/–1.2	27/1
5 8 57	23/1.2	4/–1	24/0.8
No. 2	23/0.6	4/–1.2	26/1.6
5 9 57	19/0.2	–1/–2.6	25/0.8
5 10 57	20/1	–1/–2	31/1
	14/1	0/–2	14/1
	13/0.4	0/–2	16/1

The effect of rapid bleeding upon left ventricular pressure is given in Table III. The volumes of blood removed were from 360 to 700 c.c., and the time required for bleeding was 89 to 236 seconds. Control left ventricular diastolic pressures were zero to 5 mm. Hg. During bleeding, maximum negative diastolic pressures were –2.5 to –6.5 mm. Hg. N [illegible]

Table I. Effect of Obstruction to Inflow Upon Left Ventricular Pressure, mm. Hg

DOG		BEFORE OBSTRUCTION	DURING OBSTRUCTION	FOLLOWING OBSTRUCTION
1-11-57		92/5	10/–3.5	86/5.5
		95/3	7/–5.5	73/3.5
		86/1.5	8/–3.5	93/3
	(N.E. inf.)*	138^{+}/0	48/–8.5	138^{+}/6
1-18-57		63/1.5	17/–3	73/1.5
		50/0	13/–5	73/3
1-25-57		116/6	–5/–10	118/2.5
		116/3	10/–5	115/7.5
	(N.E. inf.)	165/4	–10/–20	185/0
	(N.E. inf.)	168/8.5	14/–6	150/8
	(N.E. inf.)	167/6.5	9/–6	170/15
		110/0	15/–8	105/0
2-15-57		115/1.5	20/–2.5	150/2.5
		124/6.5	15/–5	130/2.5
2-22-57		64/3.5	9/–2	--
		104/1.5	5/–3.5	88/3.5
	(N.E. inf.)	150/3.5	23/–10	103/6.5
		106/3.5	20/–18	96/3.5
	(N.E. inf.)	146/1.5	6/–17	152^{+}/4.5
3- 8-57		117/0	24/–4	125/3
3-22-57		126/2.5	10/–5	130/6
		126/6	9/–5	155/9
	(N.E. inf.)	205/8	25/–5	--
3-22-57	(N.E. inf.)	205/4	13/–9	--
		90/4	15/–3.5	115/4
		130/7.5	18/–3.5	118/4
4- 5-57		135/7.5	14/–5	180/10
		125/4	10/–5	157/5
	(N.E. inf.)	200/2.5	15/–5	--
4-12-57		113/5	10/–9	115/2.5
		115/2.5	0/–10	108/1.5
	(N.E. inf.)	200^{+}/0	15/–10	200^{+}/0
	(N.E. inf.)	200^{+}/2.5	20/–10	200^{+}/2.5
	(N.E. inf.)	240/5	23/–10	240/5
		110/2.5	9/–6	105/2.5
		105/1	10/–5	105/2.5
5- 6-57		91/3	8/–2.5	103/5
		88/4	9/–2.5	80/3
	(N.E. inf.)	140/8	20/–5	--
	(N.E. inf.)	145/1.5	30/–3	--
5- 8-57		70/2.5	–1/–3	60/5
No. 1		73/1	16/–2.5	74/1
	(N.E. inf.)	120/1.5	21/–2.5	--
		115/2.5	22/–2.5	--
5-9 -57		105/2.5	15/–4.5	150/2.5
5-10-57	(N.E. inf.)	125/0	15/–5	140/0
		88/2.5	12/–2.5	75/0
	(N.E. inf.)	120/0	35/–4	170/5
		80/0	8/–5	95/0
	(N.E. inf.)	150/1	13/–6	160/5

*Norepinephrine infusion.

from zero to 7.5 mm. Hg. During inflation of the left atrial balloon, negative left ventricular diastolic pressure was observed in each instance, pressures ranging from –2.5 to –18 mm. Hg. Following release of the obstruction, left ventricular diastolic pressure became atmospheric or positive in each instance, ranging from zero to 10 mm. Hg (Table I). Diastolic left ventricular pressures during l-norepinephrine infusion were zero to 8.5 mm. Hg. Upon obstructing inflow during l-norepinephrine infusion, left ventricular diastolic was small The magnitude of negative pressure developed was much less than in the left ventricle, which could be attributed to differences in pressure volume curves of the two ventricles, or to less complete emptying of the right ventricle upon inflow occlusion Brecher and Kissen[5] demonstrated that the sucking force of the left ventricle of the dog s heart was greater than that of the right ventricle

The development of negative diastolic pressure in the left ventricle during rapid bleeding is of interest Although the importance of ventricular suction in ventricular filling in the normal circulation has not been proved, this observation suggests that ventricular suction may be important in ventricular filling during states of acute hypovolemia The measurement of ventricular volume under these circumstances would be of interest, since in the active circulation, negative diastolic pressure may develop at a higher ventricular volume than was found by Brecher and Kissen in their study[5] upon the dog s heart in standstill

SUMMARY

A study was made of the effect of obstruction to inflow upon diastolic pressure in the left and right ventricles in dogs The effect of rapid bleeding upon left ventricular diastolic pressure was also observed During obstruction of the mitral valve orifice, negative pressure of -2 5 to -18 mm Hg was observed in the left ventricle In the right ventricle, diastolic pressure during obstruction to inflow was -1 to -2 6 mm Hg During acute bleeding, diastolic pressure in the left ventricle became negative, with maximum values being -2 5 to -6 5 mm Hg

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appeared as early as after removal of 100 c.c. of blood, or as late as after removal of 400 c.c. of blood. In some instances, the maximum negativity declined toward the end of the bleeding period. An example of the effect of bleeding on left ventricular pressure is shown in Fig. 3.

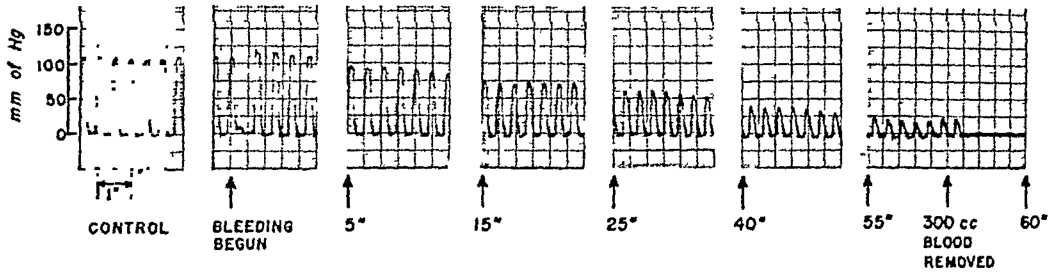


Fig. 3.—Record of left ventricular pressure during rapid bleeding from the right ventricle. Pressure is 110/0 mm. Hg at onset of bleeding and 20/–5 mm. Hg after removal of 300 c.c. blood. Horizontal tracing at end of record represents check for electrical drift.

TABLE III. EFFECT OF EXSANGUINATION UPON LEFT VENTRICULAR PRESSURES, MM. HG

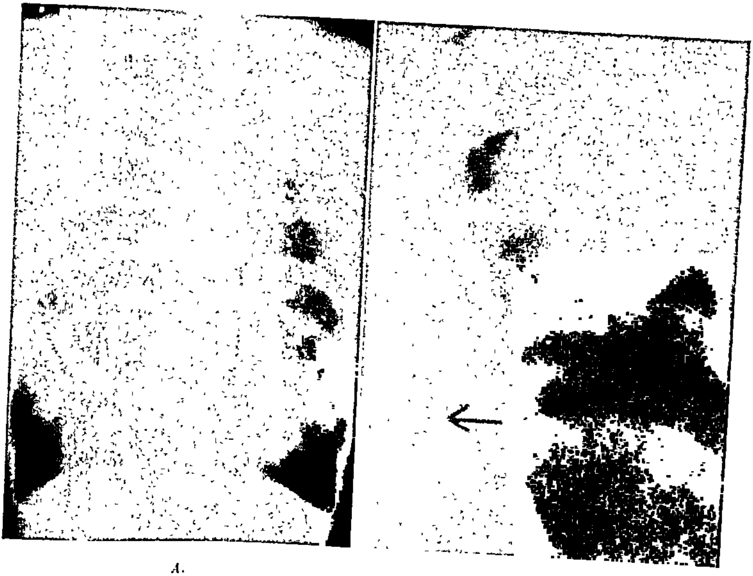
DOG	BEFORE EXSANGUINATION	DURING EXSANGUINATION
	PRESSURE	LOWEST DIASTOLIC PRESSURE
2-12-57	105/0	5/–6.5
2-22-57	137/3	8/–3.5
3-22-57	125/4	2.5/–5
5- 6-57	80/5	10/–6
5-10-57	60/0.5	8/–2.5

DISCUSSION

The development of negative diastolic pressure in the left ventricle during acute obstruction to inflow was a consistent finding, and confirms the observations of previous investigators.[1, 2, 3] When obstruction to inflow was produced during l-norepinephrine infusion, there was an increase in the amount of negative left ventricular diastolic pressure in some but not in all observations. These observations were similar to those of Brecher[3] after epinephrine, which was followed by increased negative ventricular diastolic pressure during inflow obstruction in some but not in all instances. If l-norepinephrine infusion decreases the volume of blood in the heart, then greater negative pressure in diastole may be expected, as shown by the studies of Brecher and Kissen,[5] who found increasing degrees of negative pressure when ventricular volume was decreased below a certain critical level. The interference with the circulation of l-norepinephrine to the myocardium during the period of occlusion of inflow may account for our failure to observe consistent increases in ventricular diastolic negative pressure during l-norepinephrine infusion. As observed by Brecher,[3] negative ventricular diastolic pressure did not develop immediately upon obstruction to filling, but rather after several heartbeats. This suggests that negative pressure is evidenced only after the heart has reduced itself to a critical volume by expulsion of its contents in systole.

The development of negative pressure in the right ventricle during occlusion to inflow was also consistent, although the number of animals studied of short duration, usually only several hours, and subsided spontaneously. In June, 1956, the diplopia recurred and persisted. She consulted an ophthalmologist who diagnosed an extraocular muscle paresis with strabismus. The optic fundi were normal. A complete study elsewhere, including roentgenograms of the skull and gastrointestinal tract, was negative except for a "mass in the left upper lobe of the lung." Neurologic examination showed only one internal strabismus, with normal spinal fluid. Bronchoscopy was reported negative. There were no symptoms referable to the chest.

Physical examination yielded only the following positive findings: the right pupil was slightly larger than the left; there was a slight internal strabismus of the right eye; the teeth were in very poor repair.



A.

B.

Fig. 1.—*A*, Posteroanterior films of chest showing lesion of left upper lobe. *B*, Spot film of left apex showing appearance of air crescent with change of position of patient.

Laboratory studies showed a normal urine; white blood cells numbered 14,050 per c.c. of which 72 per cent were segmented neutrophils, and 14 per cent were lymphocytes. The NPN was 42 mg. per cent.

Bronchoscopy showed a normal tracheobronchial tree. A culture of material aspirated from the bronchi yielded a heavy growth of aspergillus. Gastric contents and sputum cultures were negative for tubercle bacilli.

Roentgenograms showed a normal heart and mediastinum with minimal calcified areas in the right apex interpreted as old healed calcified tuberculosis. There was an increased density in the left upper lobe which appeared to be a cavity almost completely filled with a solid homogeneous substance. This material appeared not to be fixed in place since it changed position in the upright and decubitus positions (Fig. 1). A diagnosis of aspergilloma was made by the radiologist since it resembled so closely the cases reported by Höffke [illegible] vin.[5] Culture of material aspirated fro[illegible] corroborated [illegible] ssion.

PULMONARY ASPERGILLOMA

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CASES of pulmonary aspergillosis have been described intermittently in medical literature since the original description by Virchow[1] in 1856. He demonstrated the pathogenicity of aspergilli in the pigeon feeders of Paris, who masticated grain and then force-fed the birds. It was also described in wigmakers who employed corn meal to clean wigs. In both instances the grain was said to be the source of the mold.

Yesner and Hurwitz[1] have differentiated three forms of aspergillus pulmonary infection in man: (1) the severe pneumonic type characterized by acute pneumonia, and generalized aspergillosis which is usually fatal; (2) a mild form characterized by an acute tracheobronchitis, which runs a benign course; and (3) chronic forms which resemble tuberculosis, often with cavitation or chronic granulomas. These authors reported a case treated by left lower lobectomy in which there was a cavity, 4 by 2.5 cm. in size. Organisms were cultured from the walls of the cavity.

Pesle and Monod[2] described 3 patients who were operated upon for "aspergilloma." In 2 patients, cavity drainage was established with one death, and in one instance a successful segmental resection was performed. The lesions in these patients were similar and characteristic in that they had the appearance of a cavity, not entirely filled, in which the central opaque material surrounded by a crescent of air. The lung surrounding this lesion is usually normal.

A recent study by Vellios and associates[6] includes a case report of a patient with an "aspergilloma," in whom the radiologic and pathologic features were typical. These authors succeeded in finding 22 previously reported similar cases. They set forth a theory of origin of these lesions, which we had postulated independently that these masses occur in previously existing pulmonary lesions, such as congenital cysts, areas of bronchiectasis, old tuberculous cavities, or cavities of pyogenic origin.

The following case is presented because of the characteristic roentgen appearance which made the diagnosis immediately apparent, and because the patient had an extraocular sixth nerve palsy, causing diplopia which subsided shortly after resection of the pulmonary lesion.

CASE REPORT

Mrs. G. M., aged 52, was admitted to Milwaukee Hospital on Aug. 6, 1956, and discharged on Sept. 6, 1956. In May, 1956, she noted double vision on two occasions. The diplopia was

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that the severe pyorrhea and dental sepsis may have been the source of the aspergillosis, but proof is lacking

SUMMARY

A case of pulmonary aspergilloma is presented which was so similar in roentgenographic appearance to 7 previously reported cases that the diagnosis was made on roentgenograms of the chest alone, corroborated by bacteriologic studies

Cure was effected by lobectomy and segmental resection

A transient diplopia due to a right sixth nerve paresis present for several months before operation, has not recurred since operation

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Left thoracotomy was performed on Aug. 9, 1956. A large thick-walled abscess, 6 cm. in diameter, occupied the apical and posterior segment of the upper lobe, involving also the superior portion of the fissure between it and the superior segment of the lower lobe. The abscess was densely adherent to the chest wall and ruptured while being freed. A large amount of greenish material was removed from the abscess cavity which the pathologist reported as containing "mycelia." Upper lobectomy was deemed advisable because of small abscesses in other portions of the upper lobe. The involved superior segment of the lower lobe was also resected. The thorax was thoroughly irrigated with normal saline solution because of the contamination resulting from rupture of the abscess.

Pathologic Report.—A cavity 6 cm. in diameter contained greenish curdled material. The walls of the cavity measured up to 5-6 mm. in thickness. Part of the cyst was lined with very rough hemorrhagic pulmonary tissue, partly by smooth white pulmonary tissue, and partly by smooth white membranous tissue. There was a patent bronchus 3 to 4 mm. in diameter communicating with the cavity. The remainder of the lobe contained one small abscess, and the bronchi showed bronchiectasis of moderate degree.

Microscopically, the cyst wall was lined by mature respiratory epithelium and in some places had undergone metaplasia to resemble squamous epithelium of the skin. There was plasma-cell infiltration and considerable fibrosis.

Aspergillus was again cultured from the amorphous mass in the cavity.

Postoperatively, one gram of streptomycin, and 400,000 units of penicillin were administered once daily for 6 days. Convalescence was uncomplicated. On the fourth postoperative day, the internal strabismus disappeared completely and has not recurred. There was minimal air leak from the raw area of lower lobe in the area of local resection for a period of one week. This sealed off spontaneously.

The patient was discharged from the hospital on Sept. 6, 1956. Examinations have been conducted at monthly intervals and they have been negative. Cultures of sputum and gastric aspirations have been negative for aspergillus and tubercle bacilli.

DISCUSSION

Höffken felt that the cavity in which the mold had grown in his patient represented a congenital cyst secondarily infected. Pesle (2) suggested that the disease occurred in a pre-existing cavity, probably of tuberculous origin. It appears likely that the cavity in the present case may have been tuberculous in origin, but this cannot be definitely proved since neither history, examination of the lung, nor bacteriologic studies of the sputum, bronchial, and gastric secretions yielded evidence of tuberculosis. The presence of bronchiectasis in the resected lobe raises the possibility that the original lesion was a bronchiectatic abscess which became secondarily infected by the fungus. It seems unlikely that the fungus was the causative agent of cavitation since this is unprecedented and uncharacteristic of the usual behavior of the organism.[6] The characteristic roentgenographic appearance of a solid homogeneous mass, lying free in a cavity and surrounded by a clear halo which shifted on the change of position of the patient, seems to be pathognomonic of this form of pulmonary aspergillosis. It is significant that the lesion occurred, as in all previous cases, in the upper lobe and superior segment of the lower lobe.[5]

The extraocular muscle paresis cannot be satisfactorily explained even on the basis of a toxic reaction to the organism, but symptoms referable to vision have not occurred since operation.

The source of infection in this patient is unknown. She has always been an urban dweller and has had no contact with grain or livestock. It may be

Physical Examination—The blood pressure was 110/70 mm Hg, pulse 80, and respirations 18 The patient was a well developed but thin male The pupils did not react to light, and were round and regular but unequal in size The trachea was in the midline, and percussion and auscultation of the chest revealed no abnormalities The remainder of the general physical examination was negative Deep tendon reflexes in the lower extremities were absent

Laboratory Data—Urinalysis was negative except for microscopic hematuria The hemogram was normal and serum chemical determinations, including blood urea nitrogen, total serum protein albumin globulin ratio, carbon dioxide combining power, chlorides, Bromsulphalein, and prothrombin time, were all within normal limits Serologic test for syphilis was non reactive Roentgenographic examinations of the chest revealed the cavity to be decreased in size, however, a fluid level was still present Electrocardiogram was normal

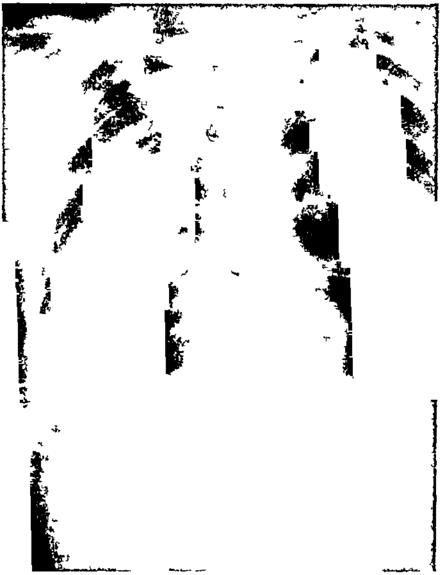

Fig 1—Roentgenogram of chest of patient on admission to hospital Large cavity in the right apex of lung

Course—An exploratory thoracotomy was performed under endotracheal gas, oxygen, and ether anesthesia through the bed of the right fifth rib Several thin adhesions binding the right upper lobe to the parietal pleura were divided without difficulty The apex of the right upper lobe was firmly attached to the parietal pleura and this was dissected free Grossly, the inflammatory process appeared to involve the major portion of the right upper lobe, as most of the lobe was noncrepitant A right upper lobectomy was performed The postoperative course was uneventful, the thoracotomy tubes were removed on the third postoperative day and the skin sutures were removed on the seventh postoperative day There was good expansion of the right middle and lower lobes and wound healing was excellent The patient has been followed in the clinic 18 months and no other evidence of pulmonary infection has been demonstrated A urine culture was negative for Aspergillus The postoperative course has been quite satisfactory and the patient has now returned to gainful em [illegible]

PULMONARY ASPERGILLOSIS

A Case Report

J. H. Smith Foushee, M.D., and Franklin G. Norris, M.D.
Winston-Salem, N. C.

ASPERGILLOSIS may affect the lung as a primary or secondary infection. It may be secondary to tuberculosis, bronchiectasis, or other chronic lung diseases, and is commonly found in post-mortem material. However, primary pulmonary aspergillosis is not a common clinical entity. Reports are now beginning to appear in the literature which help to elucidate some frequent findings in this entity. It is only recently that pulmonary resection was undertaken for this lesion. As increased numbers of cases appear in the literature, a more thorough evaluation as to etiology, symptomatology, physical findings, roentgenograms, and therapy may be obtained. The case to be presented represents a patient with primary pulmonary aspergillosis who underwent surgical resection of the involved lobe of the lung, with apparent cure of his disease.

CASE REPORT

A 44-year-old married male furniture employee was hospitalized in January, 1956, for evaluation of painless hematuria. The first episode of hematuria had occurred 2 months previously and had been followed by several recurrences. A routine urologic evaluation was performed, including intravenous pyelograms, urine cultures, cystoscopy, and retrograde pyelograms. All studies were unremarkable except for a Class III Papanicolaou smear from the right ureter. Urine cultures were negative for acid-fast bacilli; bleeding, clotting, prothrombin time, and platelet counts were normal. Roentgen examination of the chest revealed a large cavity in the right apex measuring 5.0 by 5.5 cm. in diameter with a fluid level. An area of increased density surrounded the cavity and was interpreted as representing an active inflammatory process.

The patient was transferred to a sanatorium for further diagnostic studies. Five sputum examinations, 3 gastric lavages, and 3 urine specimens were all negative on direct smear and culture for acid-fast bacilli. Intradermal skin tests for tuberculosis, blastomycosis, coccidioidomycosis, and histoplasmosis were nonreactive. A bronchogram, using Dionosil, revealed no abnormalities; the contrast medium did not enter the cavity in the right apex.

The patient was readmitted to the hospital in February of 1956 for elective pulmonary surgery.

Past history included gonorrhea and syphilis infection, in 1931, treated with salvarsan; repeated urethral dilations for stricture; penicillin treatment on the basis of positive serum and spinal fluid Wassermann in 1947 and 1950; bismuth and tryparsamide therapy in 1948; duodenal ulcer with obstruction and subtotal gastrectomy in 1954.

From the Departments of Pathology and Surgery, The Bowman Gray School of Medicine of Wake Forest College and The Laboratories of Pathology of The Baptist Hospital, Winston-Salem, N. C.

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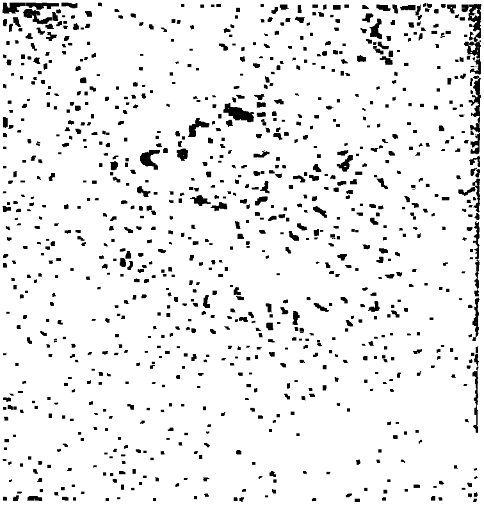

Fig. 3.—Photomicrograph of a granuloma in lung adjacent to a cavity. Giant cells, epithelioid cells, and granulocytes are present (×142; *reduced ⅙*).

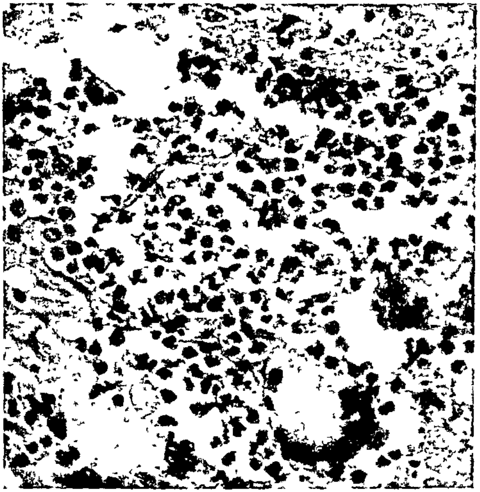

Fig. 4.—Higher magnification of Fig. 3. Note the hyphae of aspergillus and the giant cells. (×180; reduced ⅙.)

Pathology.—

Gross: A lobe of a lung that weighed 200 Gm. The surface was gray, mottled black, and red, and a number of small blebs were present. On sectioning, a cavity measuring 3.0 cm. was seen near the hilus. The cavity was filled with yellow, soft material and the wall was 0.1 to 0.4 cm. in thickness. Adjacent to the large cavity were many smaller cavities that varied from 0.1 to 1.0 cm. Many of these were filled with yellow, soft material. The bronchus was slightly dilated but did not communicate with the cavities.

Microscopic: The lung contained numerous cavities that were lined by epithelioid cells, multinucleated giant cells, and fibrous tissue. In their centers were necrotic material, a large number of granulocytes, macrophages, and numerous branching hyphae. Similar hyphae were present in the wall, surrounded by multinucleated giant cells. These hyphae were periodic acid-Schiff positive. In the surrounding lung tissue there was rather marked fibrosis as well as an increased number of lymphocytes. Areas of focal calcification as well as scattered macrophages containing brown pigment were present in the fibrous tissue. The bronchi and bronchioles were slightly dilated and contained lymphocytes and plasma cells in their walls.

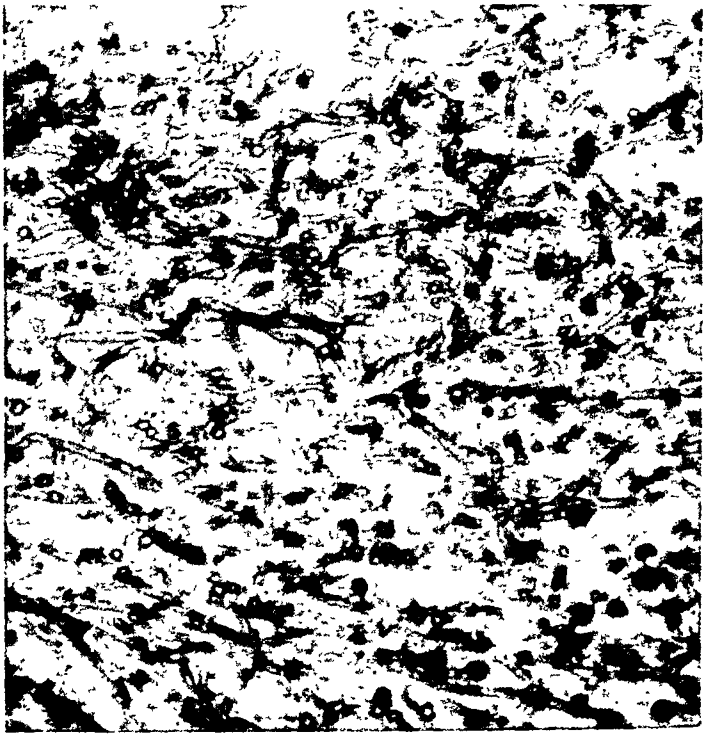

Fig. 2.—Photomicrograph of hyphae of aspergillus in center of large cavity in lung. (×480; reduced ⅙.)

Bacteriology: The resected lung was cultured and a pure culture of *Aspergillus fumigatus* Fresenius was obtained. The fungus was identified by Dr. Kenneth B. Raper, Department of Bacteriology, College of Agriculture, University of Wisconsin, Madison, Wisconsin.

Acid-fast cultures were negative for *Myocbacterium tuberculosis.*

DISCUSSION

Aspergillus is commonly found in nature and its spores frequently infest both plants and animals. Most of the cases of aspergillosis of the lungs in human beings have been due to the species *Aspergillus fumigatus.* This is the organism which was cultured from our patient. Conant and associates[1] state that the

Microscopically, Aspergillus may produce a nonspecific inflammatory reaction or a chronic granulomatous response, the latter being defined as the proliferation of reticulo-endothelial cells or macrophages in which granules or nodules are formed. In this case there was a granulomatous reaction with multinucleated cells present. From the experimental work of Cooper,[2] it may be inferred that the fungus first produces a nonspecific inflammatory reaction which later may become granulomatous if enough time elapses. In Cooper's experiments with rabbits, 6 days were required for the lesions to become granulomatous.

The fungus is seen in the tissue as branching septate hyphae. In our patient, colonies of the fungus were found in the cavity as well as hyphae in the wall of the cavity, with a cellular reaction around them. This latter finding, we feel, eliminates the possibility that these organisms might have been a contaminant. To definitely establish the diagnosis of aspergillosis, the fungus must be cultured from the lesion and a reaction to the hyphae must be seen in the tissue. As pointed out by Hughes, this fungus in the tissue has to be distinguished from Mucor and Candida.

Treatment of this disease in the lung would seem to depend on whether the disease is localized to a lobe or an entire lung, or whether both lungs are involved. Van Ordstrand's patient, in which there was bilateral pulmonary infiltration without cavity formation, was treated with iodine with apparently good results. In this case and those of Hockberg, Gerstl, Hertzoz, Yesner, Hughes, Friedman, and Vellios, removal of a localized lesion by lobectomy or lobectomy with segmental resection proved a very satisfactory method of treatment. Conant states that iodine therapy should be given after the surgical procedure. However, before conclusions can be drawn concerning treatment, more experience will be necessary.

SUMMARY

The clinical, roentgenographic, and pathologic findings and treatment of a case of primary pulmonary aspergillosis have been reported and discussed. This disease should be considered in any patient with localized or diffuse pulmonary lesions with or without symptoms.

This case is the eighth case of localized primary pulmonary aspergillosis treated by lobectomy reported in the United States to date.

We wish to thank the Department of Bacteriology, Bowman Gray School of Medicine, for its cooperation and Dr. Kenneth B. Raper, Department of Bacteriology, College of Agriculture, University of Wisconsin for identifying the fungus. The photographs and photomicrographs were prepared by Messrs. Ben Morton and Thomas Webster.

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disease is commonly seen in the following workers: (1) squab feeders, (2) fur cleaners who use rye flour, and (3) agricultural workers exposed to dust from threshers. The patient under discussion was a furniture worker.

Earlier writers on the subject of aspergillosis had classified the disease into two types: the "superficial" involving the bronchi or trachea; and the "deep" or "ulcerative" form, involving the parenchyma of the lung. Probably better terms would be bronchial and pulmonary forms. This discussion will be confined to the latter type. Certainly, there is a great deal of overlapping between the two forms.

Patients who have pulmonary aspergillosis may or may not have symptoms when first seen by the physician. The patient under discussion and the case of Yesner and Hurwitz[11] and Vellios and his co-workers[10] had no symptoms relating to their pulmonary disease. If symptoms are present they often resemble those of chronic pulmonary tuberculosis. Hertzoz,[5] Hockberg,[7] Gerstl,[4] Friedman,[3] and Hughes[6] and their associates report patients who had one or more of the following symptoms: cough, fever, chest pain, and hemoptysis. In Hockberg's case, hemoptysis was a major problem requiring blood transfusions. The duration of symptoms in the cases reported varied from weeks to years.

Roentgenographic findings in pulmonary aspergillosis vary tremendously. In the cases of pulmonary aspergillosis reported, there was an increase in density in one or both lungs which was diffuse or localized. Van Ordstrand's[9] patient had bilateral infiltrations extending out from each hilum—a pattern which some authors have called the "sun burst" effect. This patient and those of Gerstl, Hockberg, Hughes, and Vellios had radiographic evidence of a cavity or questionable cavity present in the upper lobe of a lung.

The series reviewed and collected by Levin[8] includes 27 cases of pulmonary intracavitary "fungus ball" demonstrated radiologically. Of these, 22 were identified by culture as *Aspergillus fumigatus* and 5 as *Candida albicans*. The author described very clearly the mechanism of these roentgenologic characteristics. The fungus multiplies within the cyst, or cavity, producing an ovoid mass, and the surrounding varying densities of wall of cavity and lung parenchyma produce the pattern of a thin-walled cavity containing a central mass, often with a halo of air. This fungus ball was seen to change position in several instances. Vellios reports a similar case in which an intracavitary "fungus ball" was present. In the case reported here, the fungus mass in the cavities was interpreted as a fluid level. However, gross examination of the specimen revealed no fluid and the central mass was composed of necrotic debris and branching hyphae. Fluid levels to date have not been reported in this process.

Grossly, pulmonary aspergillosis may produce grayish-white nodules in both lungs. However, in many of the cases reported, the disease was confined to one lung or lobe, in which there was a grayish-white area of consolidation. There is often necrosis of tissue and cavity formation. The lobe of the lung in this case contained one large cavity and several smaller ones. All the patients on whom surgical resection of lung has been performed have had a cavity in the upper lobe or, much less commonly, apices of the lower lobes.

FENESTRATED CARDIOVASCULAR CLAMPS

CHARLES P. BAILEY, M.D.,* AND BENJAMIN G. MUSSER, M.D.**
PHILADELPHIA, PA.

INTRODUCTION

ONE of the most obvious and pressing needs in the performance of major cardiovascular surgery is for a type of vascular clamp which will not cut through, crush, or perforate the delicate structures involved. The clamp must hold securely without danger of slippage. If, for certain conditions, it also can provide for suturing of approximated vascular walls without the necessity for prior division of their substance, a great additional advantage is obtained. Thus, each end of a patent ductus arterious might be occluded by separate mattress suture lines before interruption of its continuity. Likewise, the base of a ventricular aneurysm might be sutured before amputating the sac.

DESCRIPTION OF CLAMPS

The first of these clamps to be devised was the fenestrated aortic clamp (Fig. 1, *A*). This clamp was patterned after the large Satinsky aortic clamp. With the Pilling† Atraugrip surface of closure, it provides secure holding ability without risk of crushing or perforating delicate vascular structures. It is used for repair of the arterial incision employed for supravalvular approach for aortic commissurotomy. The clamps are designed to be applied so as to occlude the lips of an aortic incision, without compromising blood flow through the residual lumen After completing the definitive surgery, the everted aortic lips are sutured by passing a double row of continuous No. 4-0 silk mattress sutures through the fenestrations in the blades of the clamp. Thus, a secure hemostatic closure may be effected even though only a narrow margin of the aortic wall may remain. An additional advantage is afforded in that the clamp regulates the depth of tissue through which each needle passes, assuring an even closure and minimizing the possibility of aortic tearing. The initial application of this clamp was such a success that an entire series of fenestrated clamps has been designed for other uses in cardiovascular surgery.

A fenestrated aortic clamp, in two sizes, was devised to facilitate plication of the aortic valve annulus (Fig. 1, *B1* and *B2*). This technique is effective in the treatment of aortic insufficiency when the valvular pathology consists basically of a relative or actual dilatation of the annulus. The beak of this

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This work was done during the tenure of a National Heart Institute Traineeship in Thoracic Surgery at Hahnemann Medical College and Hospital

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*Professor and Head of the Department of Thoracic Surgery, Hahnemann Medical College and Hospital of Philadelphia, Philadelphia, Pa.

**Chief of Department of Cardiovascular Surgery, Harrisburg Hospital, Harrisburg, Pa.

†George P Pilling & Son Co, Philadelphia, Pa

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to hold sutures easily. The clamp then may be removed and the suture line reinforced with a continuous running suture, assuring a double row for adequate hemostasis.

The pulmonary artery clamp was constructed with angulated jaws which close in a duckbill fashion (Fig. 1, *F*). This slotted clamp was devised for use

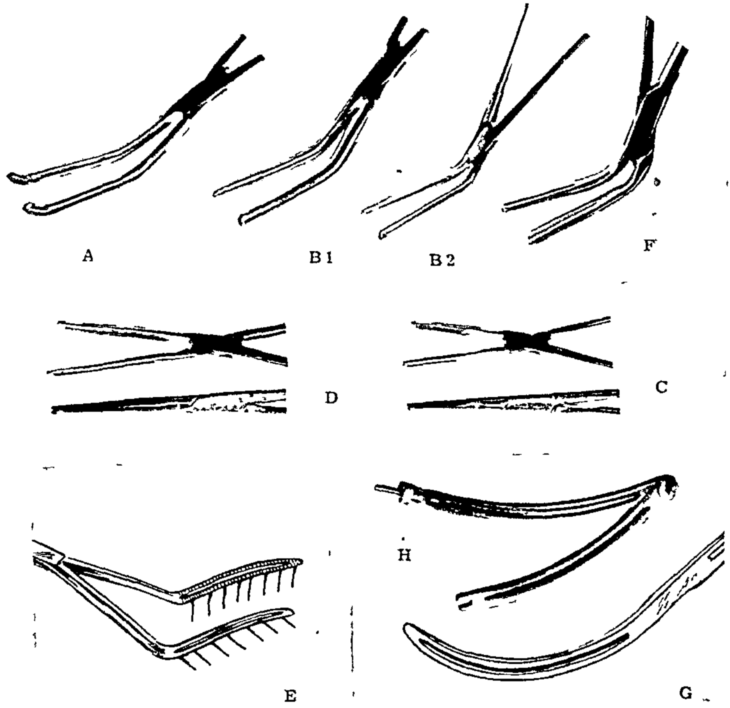

Fig 1—*A*, Aortic stenosis clamp, fenestrated This clamp is used in repair of the arterial incision used in aortic commissurotomy.

B1 and *B2*, Aortic insufficiency clamp, fenestrated. This clamp is used for plication of the base of the aorta in the correction of aortic insufficiency by cross fusion of the annulus

C, Ductus arteriosus clamp, fenestrated. The jaws are flattened and thin Before division of the ductus, each end is sutured through the fenestration Then the ductus may be divided between the two clamps without risk of possible slippage and severe hemorrhage

D, Coarctation clamp, fenestrated. The jaw of the clamp is longer and heavier than the ductus type. They provide a more certain grip upon the ends of the divided aorta.

E, Ventricular clamp, fenestrated This clamp was modified by applying teeth to the lower edge of the jaw. This allows a firm grip on the myocardium while any intraluminal clot is flushed from between the jaws of the partially opened clamp by incising the fundus of the aneurysm

F, Pulmonary clamp, fenestrated. This clamp is designed for clamping, suturing, and surgical interruption of an aortic septal defect.

G, Curved clamp, fenestrated. This clamp is designed for excision of a portion of the aorta or of the atrial wall

H, Modified Cowley aneurysm clamp, fenestrated This cl [illegible] de clamping of the base of an aortic aneurysm clamp is straight, but scalloped, allowing it to be applied to include a portion of the annulus at its tip. While the instrument conceivably might be applied in a closed type of operation, it is intended for use in the open technique, a cardiopulmonary bypass being employed during the actual surgical procedure. The right atrium is mobilized to expose the right posterolateral sinus of Valsalva. This fenestrated aortic clamp may be applied so as to plicate the portion of the annulus which subtends the posterior, noncoronary bearing cusp. This creates a functionally bicuspid valve. The insufficiency is obliterated due to the relatively greater reduction in the cross-sectional area of the aortic orifice than in the circumference of the annulus (5/9 versus 1/3). The two remaining cusps become competent by reason of the reduction in the remaining valve area. At the conclusion of the annular plication, that portion of the sinus of Valsalva and of the ascending aorta which is grasped by the blade of the diagonally reapplied clamp is sutured through the fenestrations in a continuous mattress fashion. This extension or elongation of the zone of narrowing reduces the diameter of the aorta for some distance above the valve and relieves to some degree the tension which otherwise necessarily would be borne entirely by the sutures in the annulus itself.

Fenestrated ductus arteriosus clamps, in two sizes, have been designed to permit suture closure of the ends of this structure prior to actual division (Fig. 1, *C*). The gripping surface of these clamps is made narrower to facilitate their use in children and in patients with a short ductus arteriosus. When division has been decided upon, the ductal lumen is obliterated by sutures placed through each of two slotted clamps. The ductus then may be divided between the clamps without the risk of possible slippage and severe hemorrhage. The clamps are removed separately from the ductal ends, permitting methodical oversewing and additional sutures should stitch hole bleeding occur.

A similar type of clamp has been developed for use in coarctation of the aorta (Fig. 1, *D*). The jaws of these clamps are longer and of heavier construction than the ductus clamps. The double line of compression and the intervening slots of these clamps provide a more certain grip upon the ends of the divided aorta, do not crush or perforate the tissues, and practically preclude the possibility of slippage.

The aortic stenosis clamp has been modified as suggested by Morse for use in resection of aneurysms of the left ventricle (Fig. 1, *E*). This ventricular clamp has long angulated teeth attached to the inferior margin of the gripping surface (limb below the fenestration). The clamp is applied to the aneurysm so that when closed it will approximate the cicatrix at the base of the sac. The teeth prevent slippage during the next step. The fundus of the aneurysmal sac is incised widely with the clamp closed only partially (but with teeth securely engaged). Any contained loose clots are flushed out of the sac by the force of the next ventricular contraction. Then, the clamp is closed completely and the aneurysmal portion of the scar is excised. Heavy nonabsorbable mattress sutures are used for the repair, being placed through the fenestration in the clamp. The tissue about the base of the sac is sufficiently fibrous

LUNG EXCLUSION CLAMP[*]

A New Instrument for Wedge Resections

Theodore R Hudson, M D.
Chicago, Ill.

A CLAMP has been devised which will be helpful in performing wedge resections for isolated pulmonary nodules and for lung biopsy. In the use of *two* curved clamps to exclude the wedge of lung to be removed, there is generally a certain amount of unavoidable awkwardness in coordinating the movements of the two clamps while laying the first line of sutures. Also, there is frequently a bleeder where the points of the two clamps meet but fail to secure the amputation line completely While these difficulties are certainly not monumental, they are annoying and may be time-consuming They are eliminated by this new instrument.

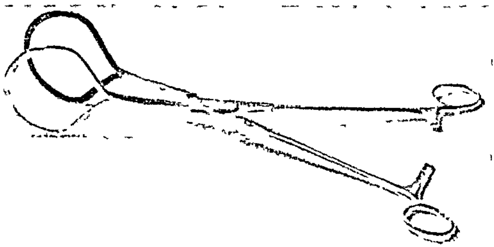

Fig 1.—Lung exclusion clamp

TECHNIQUE

The lung exclusion clamp is applied to the wedge of lung to be removed and the lock set down. The clamp has a soft action and will not mangle pulmonary tissue. A running mattress suture is taken below the clamp and close to it and tied at each end, leaving a 2-inch tail for traction at each knot. The wedge of lung is excised inside the clamp and close to it, and the instrument removed.

From the Surgical Department of Northwestern University Medical School and the Surgical Service, Chicago Wesley Memorial Hospital.

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[*] Made by George P. Pilling & Son Company, P

in aortic septal defects (aorticopulmonary fistulas). Two parallel slotted clamps are applied across the communication. Mattress sutures are passed through the fenestrations to secure intimal approximation. Both ends of the fistulous passage are thus sutured before the communication is divided. After division, the clamps are removed and the suture line is reinforced with a continuous row of No. 5-0 silk applied to the cut margins.

A curved clamp has been designed to exclude a portion of the wall of a vascular compartment such as the aorta, a great vein, or an atrium (Fig. 1, *G*). Such a clamp is useful in excision of arterial aneurysms, in the closure of tears in the atrial wall, and in partial resection of the left atrium for extensive bronchogenic carcinomas. The Atraugrip jaws assure a firm even grip while the defect is closed by placement of continuous or interrupted mattress sutures through the fenstrations.

Fenestration and an Atraugrip surface have been added to improve the Cowley aneurysm clamp (Fig. 1, *H*). The fenestration again offers the added advantage of ease of suturing with minimal chance of slippage. Firm approximation of the lips of the aneurysmal neck or base is maintained by a nut which is used to approximate the jaws of the clamp.

SUMMARY AND CONCLUSIONS

The incidence of slippage and possible laceration of the aortic wall has been reduced markedly during transarterial aortic commissurotomy by either the closed or open technique since the adoption of the fenestrated clamp. The jaws are designed with the Pilling Atraugrip surface for even nonslipping fixation of the structures grasped. This Atraugrip surface is present on both margins of the fenestration. In effect, the holding ability is that of two similar parallel clamps of the nonfenestrated type with the added significant advantage that slight extrusion of the tissue surface through the intervening slots provides. The wide gripping surface of the clamp offers secure hemostasis with less pressure upon the blood vessel wall.

A series of fenestrated clamps for use in cardiovascular surgery has been developed. These clamps offer the advantage of a double Atraugrip surface with an intervening slotlike fenestration and provide a convenient method of securing adequate hemostasis with minimum risk of tearing, crushing, or perforation of the tissues.

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

THIRTY EIGHTH ANNUAL MEETING
MAY 16, 17, AND 18, 1958
HOTEL STATLER, BOSTON, MASS

Program

MEMORANDA FOR GUIDANCE OF SPEAKERS

1 In accordance with the By Laws of the Association the papers which are read at the Meeting shall be given to the Reporter *immediately after presentation* If these papers are not submitted promptly their publication may be delayed
2 In publication it is customary to group discussions together on a series of papers Any delay in the return of corrected discussions to the Editor means that publication of all papers on the subject will be held up This is manifestly unfair to those who are conscientious in the prompt submission of their remarks for publication Undue tardiness may mean that the discussion will not be published
3 *Regular Program*—Speakers (except in symposia) will be limited to 15 minutes for presentation
4 *Thoracic Surgery Forum*—Speakers will be limted to 10 minutes for their presentations Papers on the Thoracic Surgery Forum as submitted for publication will be limited to 3,000 words At the discretion of the President and within the limits of the allotted time, one or more discussions per paper will be allowed following all formal presentations
5 *Discussion of Papers*—Discussers will be limited to 3 minutes Only members of the Association and invited speakers will have the privilege of discussing papers

MOTION PICTURE SESSION

Motion pictures will be shown on May 16, 17, and 18 Please consult official program for titles

Friday Morning, May 16, 1958

8 30 A M Business Meeting
Scientific Session REGULAR PROGRAM

Eulogy **Evarts A. Graham,** by Tom Burford

1 **Further Studies in the Surgical Management of Carcinoma of the Lung** Edward D Churchill, Richard H Sweet, J Gordon Scannell, and Earle Wilkins (by invitation), Boston, Mass
2 **Should We Insist on 'Radical Pneumonectomy' as a Routine Operation for Carcinoma of the Lung?** Julian Johnson, Charles K Kirby, and William S Blakemore (by invitation), Philadelphia, Pa
3 **Results in the Treatment of Bronchogenic Carcinoma—An Analysis of 1 008 Cases.** Sol Center (by invitation), Miami, Fla , and Thomas H Burford St Louis, Mo
4 **Coronary Artery Disease After 25 Years** Claude S Beck Cleveland, Ohio
5 **The Clinical Significance of Cor Pulmonale in the Reduction of Cardiopulmonary Reserve Following Extensive Pulmonary Resection.** William E Adams, Robert W Harrison (by invitation), Edwin T Long (by invitation), and Benjamin Burrows (by invitation), Chicago, Ill

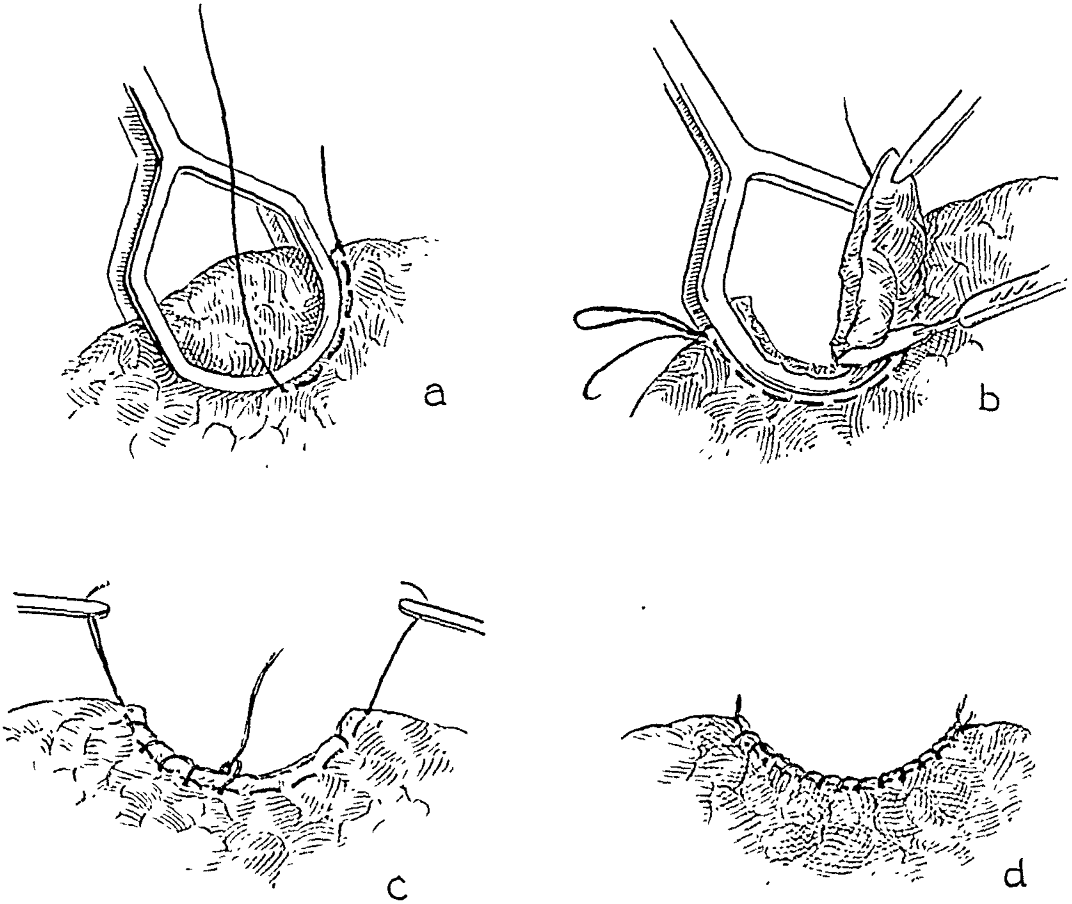

Fig. 2.—Technique for use. It is important to place the running mattress suture below the clamp before excising the specimen because the action of the instrument is so soft it will not hold the lung if the specimen is cut out first.

The lung may be held up by traction on the knot tails as the same suture is brought back to the starting point as a continuous lock-stitch, taken a bit deeper than the first suture line.

The clamp is 32 cm. in length and the opening in the jaws is 6.3 cm. across. This opening will accommodate the largest nodule one would care to resect by this method and will, of course, accept anything less, so that more than one size is not required.

19. **Elective Cardiac Arrest. The Relationship of Elevated Intracardiac Pressures to Subsequent Myocardial Function and Pathologic Pulmonary Changes.** John Ross, Jr. (by invitation), Joseph W. Gilbert, Jr. (by invitation), Edward H. Sharp (by invitation), and Andrew G. Morrow, Bethesda, Md.
20. **Lung Lymph in Experimental Pulmonary Edema.** A. Robert Cordell (by invitation), Winston-Salem, N. C., Richard A. Bahn (by invitation), Buffalo, N. Y., James C. Stephens (by invitation), Buffalo, N. Y., and H. H. Bradshaw, Winston-Salem, N. C.
21. **The Effect of Position on Pulmonary Ventilation.** Thomas F. Nealon, Jr. (by invitation), and John H. Gibbon, Jr., Philadelphia, Pa.
22. **Aortic Homograft Replacement of the Main Pulmonary Artery.** George Robinson (by invitation), Philip Glotzer (by invitation), Marvin Gilbert (by invitation), and Elliott S. Hurwitt, New York, N. Y.
23. **Experimental Results With a Prosthetic Aortic Valve.** Benson B. Roe, John W. Owsley (by invitation) and Peter C. Boudoures (by invitation), San Francisco, Calif.
24. **Post-mortem Perfusion Studies in the Evaluation of Techniques of Aortic Valvulotomy.** W. Gerald Austen (by invitation), Robert S. Shaw (by invitation), W. M. Thurlbeck (by invitation) and J. Gordon Scannell, Boston, Mass.
25. **Intercoronary Collaterals in Normal Hearts.** Sven Bellman (by invitation) and Howard A. Frank, Boston, Mass.
26. **Changes in Pulmonary Artery Pressure During Cardiopulmonary Bypass. An Experimental Study.** James B. Littlefield (by invitation), J. Francis Dammann, Jr., Phyllis R. Ingram (by invitation), and William H. Muller, Jr., Charlottesville, Va.

Saturday Afternoon, May 17, 1958

2:00 P.M. Executive Session. (Limited to Active and Senior Members.)

3:30 P.M. Scientific Session. REGULAR PROGRAM

27. **Intrathoracic Pheochromocytomas.** Herbert C. Maier, and George H. Humphreys, New York, N. Y.
28. **Management of Staphylococcic Tension Pneumatoceles by Intracavitary Suction Tube Drainage.** Elton Watkins, Jr., and Alexander C. Hering (by invitation), Boston, Mass.
29. **Topography of the Human Coronary Arteries Considered in Relation to Cardiac Surgery.** Thomas N. James (by invitation), and George E. Burch (by invitation). Sponsored by Alton Ochsner, New Orleans, La.

7:00 P.M. BANQUET AND DANCING, Hotel Statler

Attendance limited to Members of the Association and their ladies, invited speakers and their ladies.

Dinner dress preferred.

Sunday Morning, May 18, 1958

8:30 A.M. Scientific Session. REGULAR PROGRAM

30. **Open Operation for Mitral Insufficiency.** Donald B. Effler, Laurence K. Groves, William V. Martinez (by invitation), and Willem J. Kolff (by invitation), Cleveland, Ohio
31. **Direct Vision Correction of Mitral Regurgitation.** Earle B. Kay, and Cid Nogueira (by invitation), Cleveland, Ohio
32. **Satisfactory Surgical Correction of Pectus Excavatum Deformity in Childhood—A Limited Opportunity.** Kenneth J. Welch (by invitation), Boston, Mass.

6. **Effect of Severe Unilateral Hypoxia on the Partition of Pulmonary Blood Flow in Man.** Aaron Himmelstein, P. Harris (by invitation), H. W. Fritts, Jr. (by invitation), and Andre Cournand, New York, N. Y.

Friday Afternoon, May 16, 1958

2:00 P.M. Scientific Session: REGULAR PROGRAM

Address by the President, Brian Blades, Washington, D. C.

7. **Surgical Treatment of "Atypical" Patent Ductus Arteriosus.** William P. Young (by invitation), George G. Rowe (by invitation), Anthony R. Curreri, and Joseph W. Gale, Madison, Wis.
8. **Intrathoracic Aneurysms of the Aorta—Analysis of 160 Cases Treated by Resection.** Michael E. DeBakey, Denton A. Cooley, E. Stanley Crawford (by invitation), and George C. Morris, Jr. (by invitation), Houston, Texas.
9. **Experiences With the Davila-Glover Purse-String in the Correction of Mitral Insufficiency: A Critical Appraisal.** Edward M. Kent, William B. Ford, John F. Neville, Jr. (by invitation), and Don L. Fisher (by invitation), Pittsburgh, Pa.
10. **Technical Considerations in Decortication for the Pleural Complications of Pulmonary Tuberculosis.** Paul C. Samson, Duane L. Merrill (by invitation), David J. Dugan, Oakland, Calif., E. J. Shabart (by invitation), Livermore, Calif., Louis Barber (by invitation), Stockton, Calif., and James Yee (by invitation), Oakland, Calif.
11. **Achalasia of the Esophagus—Further Thoughts on Surgical Management.** William M. Tuttle, Detroit, Mich., and Robert T. Crowley (by invitation), Williamson, W. Va.

Saturday Morning, May 17, 1958

8:30 A.M. Scientific Session: THORACIC SURGERY FORUM.

12. **The Simplified Stationary Screen Pump-Oxygenator.** Jerome Harold Kay (by invitation), and Robert M. Anderson (by invitation), Los Angeles, Calif.
13. **Comparison of Relative Merits of Occlusive and Nonocclusive Pumps for Open Heart Surgery.** Paul C. Hodges (by invitation), C. Walton Lillehei, Richard Cardozo (by invitation), Andre Thevenet (by invitation), Minneapolis, Minn.
14. **Cardiac Bypass Without Artificial Oxygenator.** W. T. Mustard (by invitation), W. Sapirstein (by invitation), and Denise Pav (by invitation). Sponsored by F. G. Kergin, Toronto, Canada.
15. **Monitor and Control of Blood Oxygen Tensions and pH During Total Body Perfusion.** Leland C. Clark, Jr. (by invitation), Samuel Kaplan (by invitation), Edward C. Matthews (by invitation), F. Kathryn Edwards (by invitation), and James A. Helmsworth, Cincinnati, Ohio.
16. **Experimental and Clinical Studies of Controlled Hypothermia Rapidly Produced and Corrected by a Blood Heat Exchanger During Extracorporeal Circulation.** Ivan W. Brown, Jr. (by invitation), Wirt W. Smith (by invitation), W. Glenn Young (by invitation), and W. C. Sealy, Durham, N. C.
17. **Cerebral Blood Flow and Brain Volume Changes in Extracorporeal Circulation.** Max M. Halley (by invitation), Keith Reemtsma (by invitation), Manuel Bresler (by invitation), and Oscar Creech, Jr., New Orleans, La.
18. **Clotting Deviations in Man During Cardiac Bypass—Fibrinolysis and Circulating Anticoagulant.** Kurt von Kaulla (by invitation), and Henry Swan, Denver, Colo.

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Original Communications

MAXIMAL RECONSTITUTION OF THE STENOTIC MITRAL VALVE BY NEOSTROPHINGIC MOBILIZATION (REHINGING OF THE SEPTAL LEAFLET)

Charles P. Bailey, M.D., and Teruo Hirose, M.D.
Philadelphia, Pa.

During the 9 years which have elapsed since the initial presentation[1] by one of us (C. P. B.) of the first successful modern-type operation for mitral stenosis, many logically to be expected advances have taken place in our understanding of this disease, in our medical and surgical management, and in our operative technique. Members of the medical profession, although skeptical and reluctant at first, have accepted the initially proposed procedure with what has seemed to the innovator to be unbridled and uncritical enthusiasm. This operation has been described under the names "commissurotomy,"[1] "valvulotomy,"[2] "valvuloplasty,"[3] and "valvotomy,"[4] although all of these terms have indicated an essentially identical technique. Those who have raised their voices against the widespread application of the method or its accomplishments, albeit often for reasons which subsequently have been shown to be invalid, gradually have been overwhelmed by the increasing weight of favorable medical opinion.

Today, it is the unfortunate duty of your authors to confirm the over-all conclusion of the dissenters that this is in many respects a disappointing procedure, and to suggest in its stead a more logical and more extensive operative mobilization of the mitral valve based upon a different mechanical principle which we hope and believe will overcome the disadvantages and shortcomings of the older technique. In order to avoid the possibility of confusion with the original operation for mitral stenosis, we suggest an entirely new descriptive

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Received for publication July 30, 1957.

33. **Pectus Excavatum: An Appraisal of Surgical Treatment.** Paul C. Adkins (by invitation), and Owen Gwathmey, Washington, D. C.
34. **Hypoventilation, Hypoxia, and Acidosis Occurring in the Acute Postoperative Period.** R. Maurice Hood (by invitation), and Arthur C. Beall, Jr. (by invitation), Oakland, Calif. Sponsored by Frank Gerbode, San Francisco, Calif.
35. **Clinical and Hemodynamic Evaluation of the Surgical Correction of Aortic Stenosis.** Dwight E. Harken, Harrison Black, Warren J. Taylor (by invitation), Wendell B. Thrower (by invitation), Harry S. Soroff (by invitation), Steven Lunzer (by invitation), and Roy H. Clauss (by invitation), Boston, Mass.
36. **The Surgical Treatment of Anomalous Pulmonary Veins.** Henry T. Bahnson, and Frank C. Spencer (by invitation), Baltimore, Md.

Sunday Afternoon, May 18, 1958

2:00 P.M. Scientific Session: REGULAR PROGRAM

37. **Pulmonary Resection in the Treatment of Tuberculosis—Experience With 1,700 Cases.** Raymond J. Barrett (by invitation), Richard Jankoska (by invitation), J. Claude Day, Paul V. O'Rourke, and E. J. O'Brien, Detroit, Mich.
38. **Pulmonary Infarction Complicating Segmental Resection.** John M. Salyer, and Harold N. Harrison (by invitation), Denver, Colo.
39. **Creation of a Temporary Artificial Ductus in the Surgical Correction of Ventricular Septal Defects Associated With Severe Pulmonary Hypertension: A Two-Stage Operation.** Howard D. Sirak (by invitation), and Don M. Hosier (by invitation). Sponsored by H. William Clatworthy, Jr., Columbus, Ohio.
40. **Corrective Surgery in the Presence of Far-Advanced Pulmonary Hypertension.** C. Walton Lillehei, Donald J. Ferguson (by invitation), and Richard L. Varco, Minneapolis, Minn.
41. **Surgical Treatment of the Tetralogy of Fallot by Open Intracardiac Repair.** John W. Kirklin, F. Henry Ellis, Jr., and Dwight C. McGoon (by invitation), Rochester, Minn.
42. **Atrial Septal Defect, Secundum: Observations on One Hundred Patients Treated by Open Operation.** Henry Swan, Denver, Colo., D. Hywel Davies (by invitation), and S. Gilbert Blount, Jr. (by invitation), Denver, Colo.

completely mobilized leaflets cannot become separated freely during diastole if they continue to be bound together by cross-fused chordae or by a directly adherent papillary muscle (Fig 2).

With the passage of time, certain significant modifications were added to the original operative technique. These included "flush-out" of the thrombosed appendage[8, 19] and temporary occlusion of the carotid circulation,[8, 20, 21] both techniques having been designed to lessen the incidence of serious arterial embolization. A very important innovation was the "bare-finger" method originally suggested by Husfeldt[22] The increased tactile sensibility facilitates accuracy of orientation, permits a more effective application of digital disruptive pressure, and ensures critically accurate guidance of any necessary instrumentation.

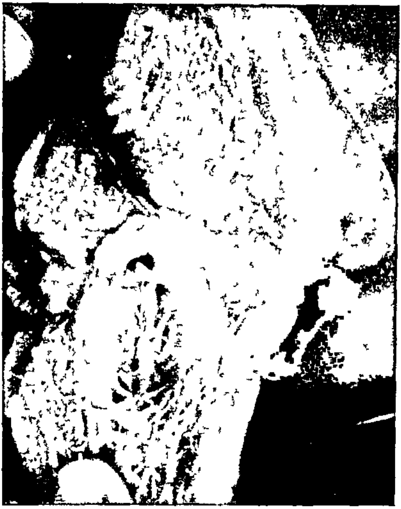


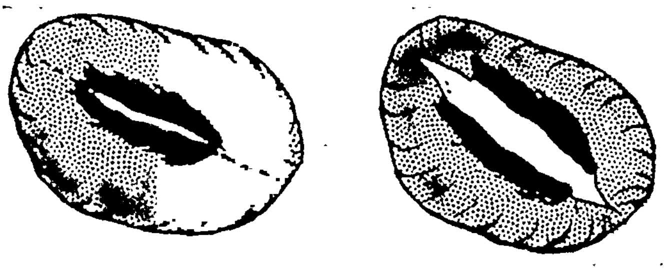
Fig 2—Strictured orifice in a heavily fibrosed valve (*From* Bailey Surgery of the Heart, ed 1, Philadelphia, 1955, Lea & Febiger, p 515)

Appreciation of the importance of alterations in the subvalvular supporting structures, as described by Brock,[4] led to the development of definitive methods for relief of any such subvalvular stenosis by Harken[23] and especially by Nichols and Jamison.[24]

The development and perfection of the right-sided approach to the mitral valve[18, 25-28] brought about a revolutionary advance in the technical facility with which the stenotic mitral valve mechanism can be mobilized At first this procedure amounted merely to a superior type of commissurotomy. It was, however, only a matter of time until the now clearly demonstrable effect of name for the procedure: neostrophingic mobilization of the septal leaflet. This is derived directly from the classical Greek: *neo*—new, and *strophings*—hinge, meaning a new hinging of the septal leaflet.

The original "corrective" operation for mitral stenosis consisted essentially of the insertion of the operator's index finger through an incision made in the tip of the purse-stringed left auricular appendage in order to palpate the stenotic mitral valve orifice, and to split apart the fused leaflets at either or both extremities of the diminutive slit or, failing in this attempt, to effect an instrumental section of the commissures by a valvulotome inserted through the appendage.[4-7] In some clinics, the valvulotome amounted merely to a ring with a cutting edge which was worn upon the finger tip.[8] Some operators have used only a dilating mechanism to accomplish an enlargement of the valve orifice.[9, 10]

In nearly all instances, the surgical principle employed has been that of separating the fused valve leaflets at the commissures, essentially a lengthening of the diminutive valve slit (Fig. 1, *A* and *B*). Appreciating that the most basic (although this admittedly is an oversimplification) action of the normal mitral valve is that of the flutter valve, cardiac surgeons were confident that they were restoring, in inverse proportion to the extent of intrinsic pathologic change, a portion of the normal-type valve action. With the valve so mobilized, little or no regurgitation is created. Since even a rather slight restoration of valvular function may suffice to bring about great clinical improvement in patients with mitral stenosis, surgeons and cardiologists alike were delighted with the immediate benefits. They were not seriously disturbed by the usual persistence of typical auscultatory evidences indicative of residual mitral stenosis.[11-13] Nor were they concerned with the likelihood of the development of recurrent mitral stenosis,[5, 14-16] although examination of typical pathologic specimens indicated that the severely stenotic mitral valve had become converted essentially into a fibrous stricture (Fig. 2).



A. *B.*

Fig. 1.—*A*, Original concept of diminutive stenotic valve slit with fibrosed (in black) margin completely surrounded by flexible valvular tissue (shaded).

B, Original concept of simple linear lengthening of the valve slit by incision or splitting at each extremity. It was felt that once the marginal tissue had been divided well through the scar and into flexible tissue, a great portion of the natural "flutter-valve" action would be restored without the creation of any regurgitation. (*From* O'Neill, Glover, and Bailey: Commissurotomy for Mitral Stenosis, J. Internat. Coll. Surg. 8: 359, Fig. 7, 1950.)

The frequent reinforcement of the primary leaflet fusion by subvalvular changes[4, 8, 17, 18] largely has been ignored although it is very evident that even the line of valve closure normally is so curved that it constitutes practically a semicircle. This ideal orifice is a far cry from the original surgical objective of simple linear elongation of the valve slit (see Fig. 1). Nevertheless, if the mutually adherent valve leaflets consistently remained thin and flexible, it would be easy enough to accept a slight modification of this concept of linear elongation, the appropriate line of separation merely being extended in arcuate fashion. It must be admitted that in certain lightly fused valves with but slight fibrous change, such a mobilization will accomplish a nearly ideal mechanical result, the original flutter valve being functionally re-established.

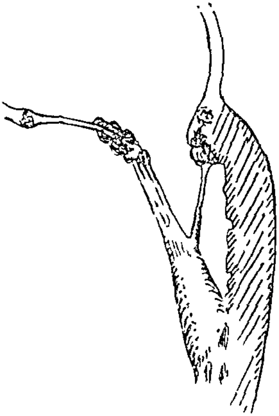


Fig 4.1 —Diagrammatic illustration of equal extents of involvement of both leaflets by the rheumatic process The distal third of the septal, but the entire apicobasal length of the mural leaflet are thickened and fibrosed. Contraction of the chordae of the third order may draw the shrunken mural cusp against the ventricular wall.

However, in actual practice, the finding of such a minimal degree of pathologic change in mitral stenosis is exceptional. Usually the entire mural leaflet is shrunken from free margin to base, is thickened and fibrosed—perhaps infiltrated with calcific salts. This involvement may extend equally for the entire annular and marginal length of the mural leaflet or it may be limited or concentrated toward one pole of the valve. Sometimes the pathologic change involves chiefly one of the original valve commissures—more often the "posterior." However, in most patients who come to surgery the majority of the length of the mural leaflet is so carnified, so shrunken, or so retracted against the ventricular wall that it is inconceivable that any worth-while mobility could be imparted to it. In all probability the maximal functional value of this altered leaflet lies in the fact that it presents an immobile ridge or shelf extending into the atrioventricular channel against which the surgically mobilized septal leaflet can abut to provide valvular competence. Undoubtedly this rigid projecting shelf impedes to some degree atrioventricular blood flow. Because of the relatively large size of the total atrio[illegible]ar passagew[illegible]is impe[illegible] of the various steps entailed in the surgery of the stenotic valve would be integrated consciously into a significantly advanced concept for maximal restoration of valvular function.

While Chiechi[29] and others[7, 18, [illegible], 30] have attempted to describe in detail the movement and function of the various components of the mitral valve mechanism, for the purpose of simplification and clear surgical understanding, the major mitral valve leaflets may be considered to act in the same manner as the two lips of a flutter valve. However, since the septal leaflet is considerably longer from free margin to basal attachment (on the annulus fibrosus) than is the mural, obviously the flutter-valve action must be an eccentric one. Further, largely because of the triangular shape of the septal leaflet (Figs. 3A and 3B),

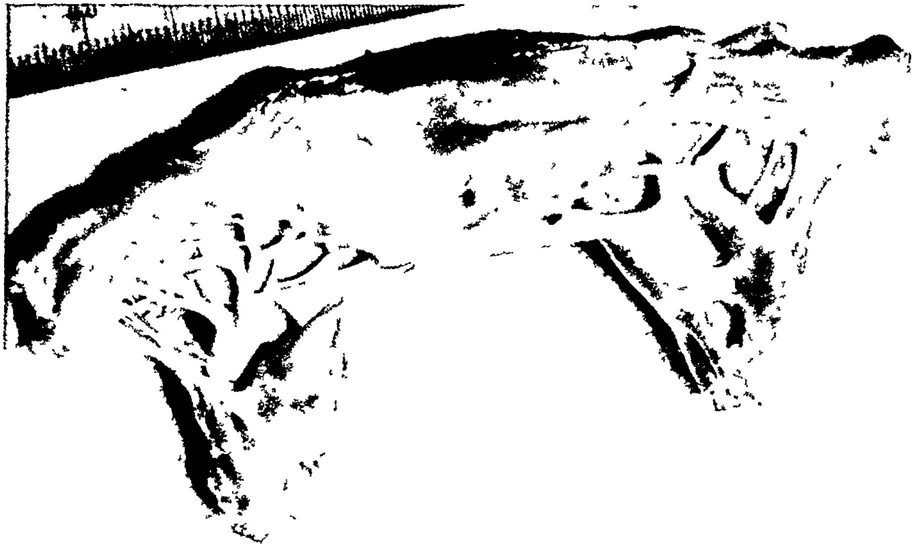


Fig. 3A.—The septal leaflet of the mitral valve being shaped like a wedge or a truncated triangle does not permit the two freely separated cusps to become approximated in a straight line. (*From* Bailey: Surgery of the Heart, ed. 1, Philadelphia, 1955, Lea & Febiger, p 492.)

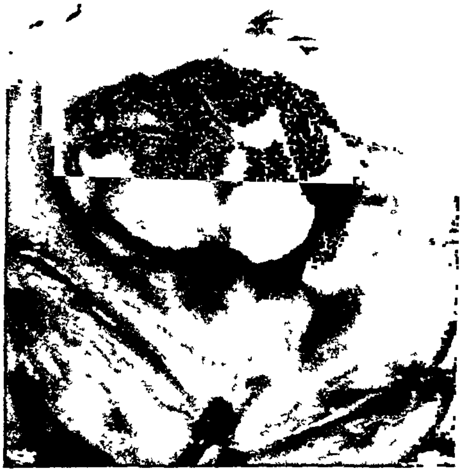


Fig. 3B.—Nearly semicircular line of closure which is characteristic of the normal mitral valve. (*From* Bailey: Surgery of the Heart, ed. 1, Philadelphia, 1955, Lea & Febiger, p 491.)

mural leaflet. When they become thickened and contracted due to the organization of inflammatory exudate accumulated upon their surfaces and within the angle formed by the junction of the ventricular aspect of the mural leaflet with the inner endocardium of the left ventricular wall, they may draw the mural leaflet toward or even against the ventricle (Fig. 4*A*). In extreme instances, this retraction may cause regurgitation because it can amount functionally to an absence of the mural leaflet over a given area. In most cases the leaflet continues to project as a rigid though diminutive shelf.

An equal extent of rheumatic involvement of both leaflets from the free margin basally will carnify (or calcify) only one third of the septal leaflet while an equivalent amount of pathologic change will render practically the entire apicobasal length of the mural leaflet completely rigid. In other words, rheumatic thickening and fibrosis of the septal leaflet nearly always is limited to the distal (marginal) third, the middle and basal thirds remaining thin, flexible, and grossly free of disease (Figs. 4*B* and 4*C*). This observation is basic to the concept of "rehinging" the septal leaflet (neostrophingic mobilization). No matter how rigid or calcified the septal leaflet may seem on first palpation, complete mobilization from the mural leaflet (and the junctional tissues and/or commissural leaflets when present) to a point at either extremity which is beyond any gross thickening or disease (usually slightly beyond the original location of the commissures) will enable the septal leaflet to bend freely upon its middle uninvolved zone unless restricted by cross-fusion or shortening of the chordae tendineae or by a direct adherence of one or both papillary muscles to the valve apex (Fig. 5, *A* and *B*). While we have recognized such direct papillary attachment in only 30 per cent of stenotic valves at the time of surgery, it is readily apparent to the examining finger that a considerable restriction in the degree of possible leaflet divergence is present at one or both poles in more than half of all operated cases (Fig. 6, *A* and *B*). Usually this is due to a shortening of the normal "yoke" of suspension created either by shortening of the chordae or by an apical lengthening of the valve leaflet due to accretion of organized exudate as described by Brock,[5] [illegible] th.

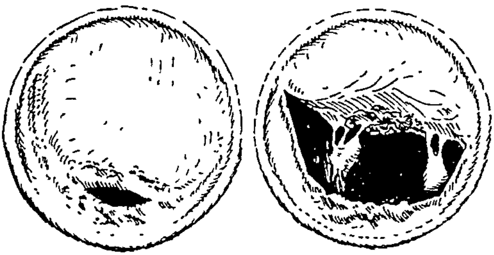

A. *B.*

Fig 5.—*A*, Diagrammatic representation of characteristic findings in severe mitral stenosis Note total fibrosis of central portion of the mural leaflet

B, Extensive mobilization of the septal cusp from the rest of the (continuous) valve ribbon so that the cleavage extends into fully flexible tissue at either pole provides free hinging upon its middle third, unless restricted by cross-fusion of the subvalvular supporting structures.

must be slight. For the sake of surgical thinking, the junctional tissues[29] may be considered to be part of the length of the mural leaflet since they are subject with it to a similar process of deformation.

Paravisini-Parra[31] has postulated that the mural leaflet of the mitral valve is more susceptible to the ravages of the rheumatic process than is the septal and it is true that extreme degrees of pathologic involvement and retraction do appear to be more frequent in this leaflet than in the septal. However, this may be more apparent than real because of anatomic differences in the respective structures of the two leaflets. The chordae of the third order attach only to the

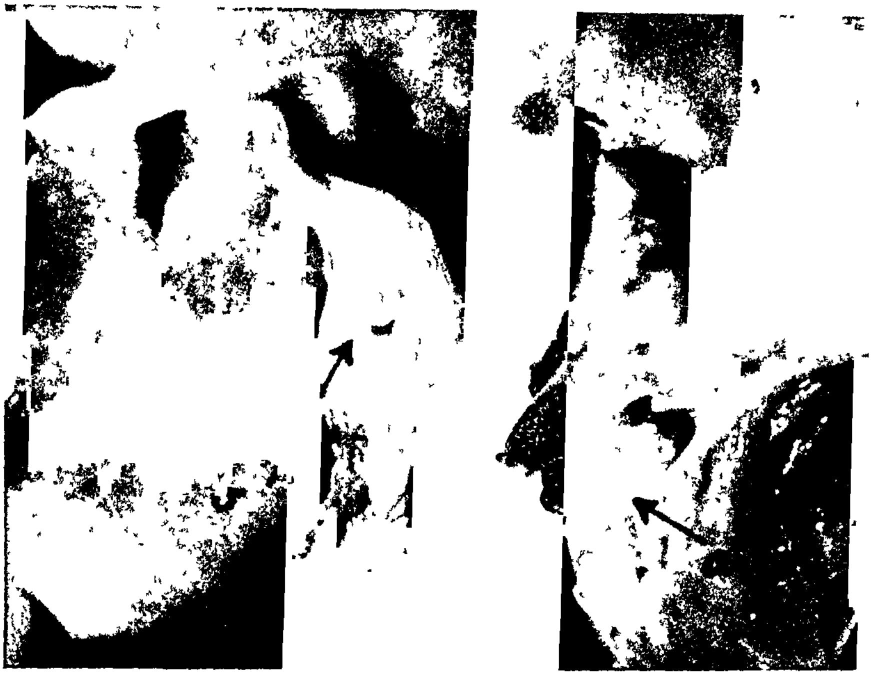


Fig. 1B.—Photograph showing retracted carnified mural leaflet (*a*) and fibrosis of the distal portion of the septal leaflet (*b*). Note thin flexible basal portion of latter.

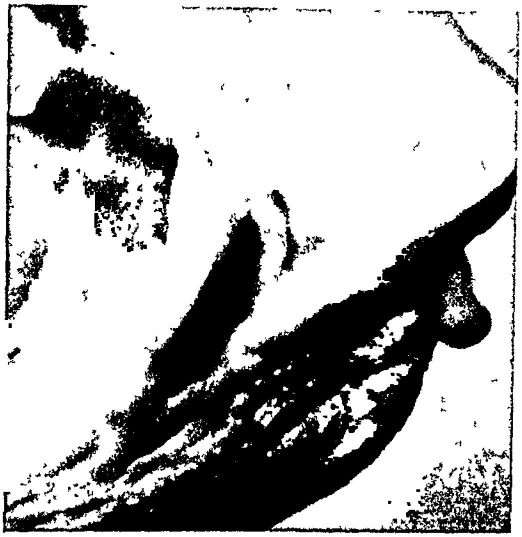


Fig. 4C.—Similar photograph of a specimen of mitral stenosis in which the mural cusp is *not* retracted but in which the characteristic disproportion in relative per cent of involvement of the respective cusps is shown. Note thin flexible proximal portion of septal leaflet.

Once the septal leaflet has been mobilized from the rigid mural leaflet and junctional tissue, the separation being extended fully to its flexible mid-portion, providing any subvalvular restriction has been eliminated, it will swing freely like a great sail in response to the hydrodynamic stresses natural to this region Its free margin diverges into the atrioventricular channel during ventricular diastole and returns during systole to abut against the passive mural ridge,

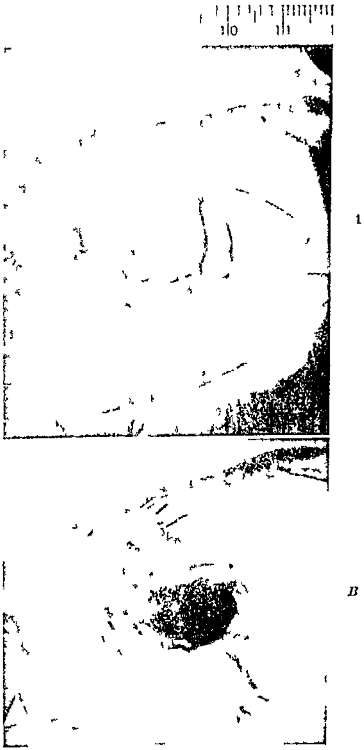


Fig 7.—A, Atrial view of stenotic mitral valve after neostrophingic mobilization (closed position)

B, Same valve in opened position Note that the fibrosed mural leaflet moves little if at all

providing competence! Thus, the "flutter valve" action of the normal bicuspid mitral valve is replaced by a "flap valve" action of the now nearly unicuspid mitral valve,[8] a characteristic mechanism in certain other vertebrate species (notably the turtle) While such modified valve action obviously is not entirely normal to the human subject, it is, an efficie tional

Only by extensive longitudinal splitting of the restricting pathologic chordopapillary complex (when present) can this binding or limitation of valve movement be abolished! In most instances, this will necessitate complete longitudinal splitting of the papillary muscle (perhaps initiated by direct incision at the "crotch" of the suspension) fully to its ventricular origin. Fortunately, such longitudinal separation of the papillary muscle does not influence its ability to support the respective leaflets and, hence, does not tend to diminish valvular competence (Fig. 6, *C* and *D*).

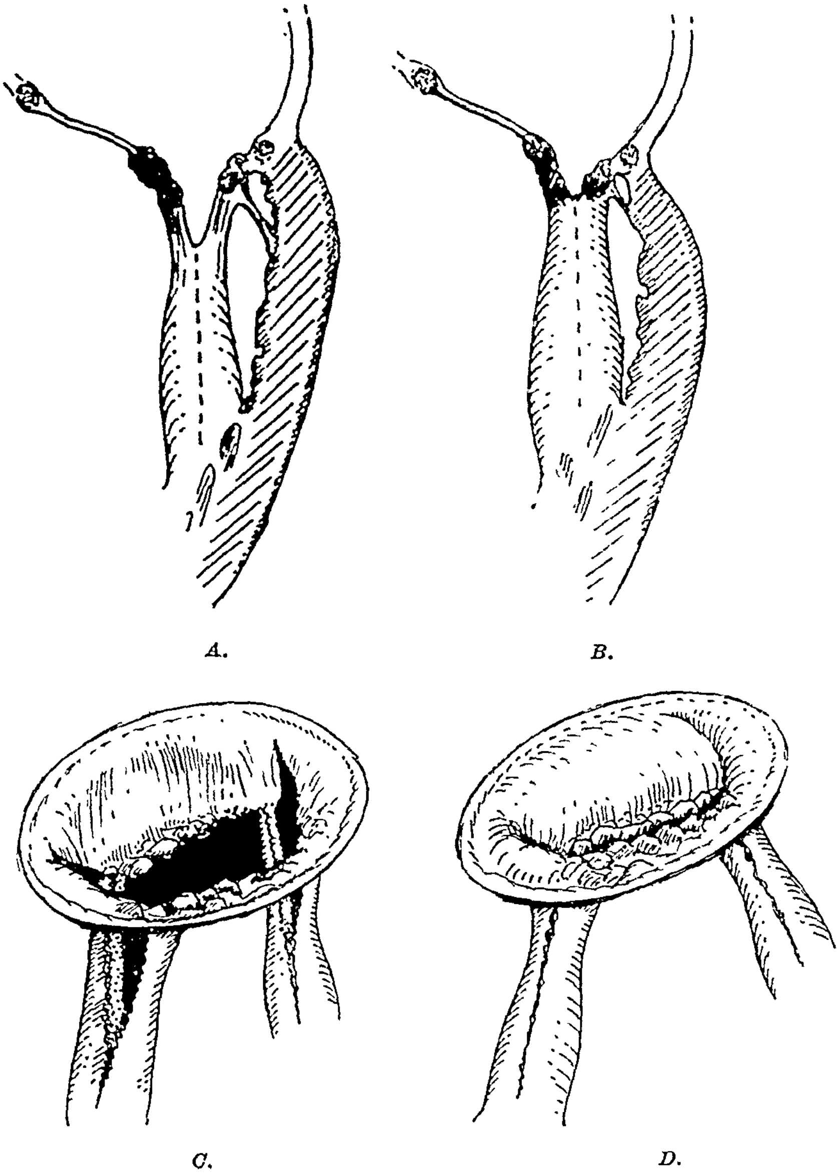


Fig. 6.—*A*, Diagrammatic illustration of shortening of the normal "yoke" of suspension of the mitral valve. This restricts possible leaflet divergence. It may be caused by actual shortening of the fibrosed chordae or by "lengthening" of the leaflets due to organization of exudate deposited upon their free margins.

B, Direct adherence of chordopapillary fusion mass to valve apex due to extreme stage of this process.

C, Complete rehinging of septal leaflet by extensive longitudinal splitting of the papillary muscles as well by total mobilization of this leaflet from the rest of the valve ribbon.

D, Even though the mural cusp is rigid and immobile, free flap-like movement of the septal leaflet provides excellent valve action and full competence.

RESULTS OF NEOSTROPHINGIC MOBILIZATION

Because of increasing dissatisfaction with even the improved commissurotomy procedure possible by the right-sided approach,[26, 27] the described new concept of a method for *complete reconstitution of valve action* was born.

In spite of the evolution of this concept of rehinging of the septal leaflet which would seem to approach the surgical ideal, we have not always succeeded in accomplishing this objective in practice. Among 227 consecutive patients who *have been operated for essentially isolated* mitral stenosis at our clinic since the deliberate application of this principle surgically for the first time on Sept. 26, 1955, it is felt that a completely satisfactory mobilization was accomplished in only 70 per cent (Table I). In 20 per cent, the technical achievement (although less than desired) was considered to be equal or superior to that which would have been produced in the same individuals by the older technique of commissurotomy. In most instances the failure was related to termination *of the mobilization prior to logical completion because of the beginning development* of a regurgitant jet at one or both commissures.

TABLE I. ESTIMATES OF ACCOMPLISHMENT IN RESTORATION OF THE MITRAL VALVE, BOTH ANATOMICALLY AND FUNCTIONALLY

Anatomic opening 100%, function 100%	50	156	70%
More than 90% of valve function restored	106		
Acceptable (over 70% of function re-established)		23	10%
Unsatisfactory results		48	20%
Limited valvular opening	24—10%		
Increased or created regurgitation	19— 8%		
Subvalvular fusion not completely relieved	5— 2%		
Total	227 Cases		

Instrumentation was employed in 181 patients (Table II).

TABLE II. PROPORTION OF DIGITAL AND INSTRUMENTAL MITRAL SEPARATION

Both commissures opened			218
Left and right knife used	100	175	
Left knife only used	53		
Right knife only used	22		
Digital only		43	
Anterior commissure only			5
Left knife used		4	
Digital only		1	
Posterior commissure only			4
Right knife used		2	
Digital only		2	
Total			227

Regurgitation of greater than 1+ degree was created (or exaggerated) in 19 patients. However, 76 patients have manifested a new systolic murmur postoperatively (Table III). Since many of these patients presented tricuspid disease, this sign is considered of uncertain significance.

Calcification of the valve which was considered severe in 34 instances did not seriously reduce the surgeon's ability to achieve a satisfactory mobilization (Tables IVA and IVB).

mechanism. Moreover, it is one which can be established by surgery in most patients with mitral stenosis, whereas a normally functioning "flutter-valve" can be restored but rarely (Fig. 7, *A* and *B*). In those relatively rare instances of very slight deformation, a similar type of surgical mobilization merely results in an exquisitely perfect normal type of bicuspid valve action since, in actuality, it then amounts to a complete renovation of the valve.

DISADVANTAGES OF PREVIOUS OPERATIONS

While the objections to the former method of commissurotomy apply with especial force to those procedures which have been performed through the disadvantageous left-sided approach, they also are pertinent in lesser degree to similar operations performed from the right. It was as much limitation of the earlier concept which restricted the surgeon's operative initiative as *the technical faults in his approach.*

The fundamental criticisms are the following:

1. Because of technical and conceptual limitations, the extent of valvular mobilization achieved by efforts at commissurotomy by the older method usually amounted to but 20 to 30 per cent of the ideal. In two thirds of the patients, but one commissure was opened.[8, 18] It is believed that the per cent of functional restoration usually has been significantly low. This has been attested by the consistent persistence of auscultatory manifestations of mitral stenosis after surgery as well as by physiologic evidences of residual obstruction at the valve level.

In many instances this residual stenosis was reinforced and aggravated by an element of subvalvular stenosis, usually unrecognized and less often relieved (now believed to be present to a significant extent in more than half of these patients).

2. Inadvertent production of significant mitral regurgitation. This was a logical and not infrequent end-result of the customary employment of the clumsy and inefficient left-sided approach and the uncertain orientation which it affords.

3. Unsatisfactory clinical course of operated patients. While it is felt that over 75 per cent of patients receive significant benefit from a mitral commissurotomy operation, in many cases this has amounted merely to prolongation of a life of invalidism rather than to the rehabilitation which an ideal correction of the mitral obstruction might have offered.

4. The increasing evidence that mitral commissurotomy is attended by a high incidence of recurrence of mitral stenosis after the elapse of several years.[16] It is believed that 20 to 40 per cent of patients operated by the older technique will suffer from restenosis and ultimately will require repetitive surgery.

5. A seemingly irreducible incidence of arterial embolization due chiefly to dislodgment of thrombotic fragments from a clotted left auricular appendage.[8, 27]

Subvalvular pathologic change was found in 128 patients (Table VA). In 70 (30.8 per cent) valves, direct papillary muscle adherence to the valve apex was recognized at one or both poles. However, complete relief of subvalvular stenosis was not accomplished in 20 cases (Table VB).

Arterial embolization was produced at surgery in 7 patients (3 per cent). In 5 patients, this was cerebral, in 2, peripheral (Table VIA). No limb amputations were required. While these accidents were believed to have been produced by inadvertent dislodgment of calcific particles from the valve at the time of surgery, it is possible in some that dislodged thrombotic material from a clotted atrium was responsible (Table VIB). However, avoidance of the left auricular appendage as an entrance route would seem to have reduced the likelihood of thrombotic embolization.

TABLE VI A. INCIDENCE OF ARTERIAL EMBOLISM IN 227 CASES OF MITRAL RECONSTITUTION

	NUMBER OF CASES	DEATHS
Cerebral embolism	5 (2.2%)	2
Peripheral embolism	2 (0.8%)	0

TABLE VI B. RECOGNIZED INCIDENCE OF THROMBOSIS OF THE LEFT ATRIUM

No thrombus felt			204
Thrombus			23 (10%)
Loose fresh thrombus		18	
Required removal	5		
Not disturbed	13		
Placenta like organized thrombus (not disturbed)		5	
Total			227

The technical results achieved are shown in Tables VIIA and VIIB.

TABLE VII A. OPERATIVE MORTALITY OF OPERATIONS FOR MITRAL STENOSIS

	NUMBER OF CASES	OPERATIVE DEATHS
Mitral commissurotomy from the left side	1,000	77 (7.7%)
Mitral commissurotomy from the right side (before Sept., 1955)	150	13 (8.6%)
Neostrophingic mobilization (from Sept., 1955 to Jan., 1957)	227	11 (4.8%)
Good technical result More than 90% valvular function restored	156	4 (2.5%)
Compromised technical result, Less than 90% valvular function restored	71	7 (10%)

TABLE VII B. THE FOLLOW UP STUDIES OF MITRAL VALVULAR RECONSTITUTION (MORE THAN 3 MONTHS)

	EXCELLENT	IMPROVED	SAME	DEATHS	TOTAL
Ideal or near ideal functional valvular opening obtained	65 (54.6%)	45 (37.8%)	5 (4.2%)	4 (3.4%)	119 (100%)
Less satisfactory valvular opening established	21 (41.2%)	22 (43.1%)	1 (2.0%)	7 (14.7%)	51 (100%)

The diastolic murmur and sharp first sound of mitral stenosis were ished (by phonocardiographic determination) in half of the en series T

TABLE III. PRE- AND POSTOPERATIVE CHANGES IN VALVE COMPETENCE

	PREOPERATIVE GRADE OF REGURGITATION	POSTOPERATIVE GRADE			
		NONE	MINOR	MODERATE	SEVERE
None	167	51	111	3	2
Minor	54		46	7	1
Moderate	6			6	
Totals	227	51	157*	16*	3*

*Seventy-six patients had a new systolic murmur postoperatively.

TABLE IV A. INCIDENCE OF CALCIFICATION OF THE VALVE

No calcification		152	(67%)
Calcified valve		75	
Minimal degree	41		
Moderate degree	23		
Heavily calcified	11		
Total		227	

TABLE IV B. INFLUENCE OF CALCIFICATION OF THE MITRAL VALVE UPON FINAL FUNCTIONAL VALVULAR OPENING

	MORE THAN 90% FUNCTIONAL OPENING OBTAINED		LESS THAN 90% FUNCTIONAL OPENING OBTAINED		TOTAL
Fibrotic noncalcified valve	110	72.4%	42	27.6%	152
Partially or heavily calcified valve	46	61.4%	29	38.6%	75
Totals	156		71		227

TABLE V A. SUBVALVULAR INVOLVEMENT

No subvalvular involvement recognized			99	(43.6%)
Anterior subvalvular changes		28		
Papillary fusion to valve	8			
Chordal agglutination	20			
Posterior subvalvular changes		76		
Papillary fusion to valve	46			
Chordal agglutination	30			
Bipolar subvalvular changes		24		
Papillary fusion to valve	16			
Chordal agglutination	8			
			128	(56.4%)
Total			227	

TABLE V B. THE RESULTS OF TREATMENT OF THE SUBVALVULAR STENOSIS IN THE CASES OF MITRAL RECONSTITUTION*

No subvalvular stenosis	99
Subvalvular changes not treated	
No effect on the valvular function	29
Minimal changes	3
Unable to separate	5
Subvalvular fusion treated	
Complete or almost complete relief obtained	64
Physiologically reasonable function obtained	12
Unsatisfactory separation accomplished	15
Total	227

*Guillotine knife or papillotome used, 12 cases; digital pressure or fingernail used, 84 cases.

It is interesting that there is no direct linear relationship between the functional restoration of the valve and the old measurement of valvular opening in "fingers."

TABLE IX. RELATIONSHIP BETWEEN ESTIMATE OF PERCENTAGE OF FUNCTION AND OPENING MEASURED IN "FINGERS" IN CASES OF NEOSTROPHINGIC MOBILIZATION

	MORE THAN 90% FUNCTIONAL OPENING		LESS THAN 90% FUNCTIONAL OPENING	
More than 2 fingers	16	10%	4	5.6%
2 fingers	86	55%	21	30%
1 and ¾ fingers	47	31.4%	29	40%
1 and ½ fingers or less	7	3.6%	17	24.4%
Totals	156	100%	71	100%

THE SURGICAL TECHNIQUE

The Thoracic Entrance.—The patient is placed in the supine position. A right anterolateral thoracic incision is made from the sternal border to the mid axillary line, curving below the breast in the female.

The pectoral muscle fibers are split laterally and incised medially to expose the fourth intercostal space. The intercostal muscles and pleura are divided extensively from the internal mammary vessels to the transverse processes of the vertebrae. The former are divided between individual ligatures. The fourth and fifth costal cartilages may be divided at their sternal junctures. The ribs are spread widely apart in gradual increments by a Finochietto type of fixed retractor in order to avoid fracture. The right lung is compressed posteriorly.

The Pericardial Incision.—The pericardial sac is incised parallel and 1.5 cm anterior to the right phrenic nerve for its entire length. The caudal extremity of this longitudinal incision may be extended anteriorly to the sternal border to obtain additional exposure.

The vessels which cross the incised portion of the pericardium are individually ligated and the lips of the incision in this sac are retracted with weighted stay sutures.

The Cardiac Incision.—The interatrial groove is easily identified by gentle upward retraction of the right atrium by the gauze covered fingers of an assistant. It will be seen, at least during certain phases of the cardiac cycle, as a distinct line or depression communicating the posterior aspects of the inferior and superior venae cavae.

The mid portion of the groove is dissected to a limited extent, usually 1 cm in length and 8 mm in depth, but with individual variations.

A purse string suture of heavy No. 2 braided nylon, swedged on a curved round edged needle, is placed widely about this dissected area. The line of encirclement should be at all points at least 1 cm distant from the line of dissection. Each bite of the suturing needle should penetrate into the corresponding atrial lumen. The external loops of the suture should be made longer than the internal ones. The free ends are incorporated within a Rumel Belmont tourniquet.

VIIIA and VIIIB). By simple auscultatory examination, the rate of apparent abolition of this murmur was much greater (Fig. 8, *A* and *B*).

TABLE VIII A. POSTOPERATIVE DISAPPEARANCE OF MITRAL DIASTOLIC MURMUR AFTER NEOSTROPHINGIC MOBILIZATION (PHONOCARDIOGRAPHIC DETERMINATION)

	NUMBER OF CASES	DISAPPEARANCE OF DIASTOLIC MURMUR	
Complete or near complete restoration of valvular function	148	75	(51%)
Incomplete restoration of valvular function	56	25	(45%)
Total	204	100	(49%)

TABLE VIII B. POSTOPERATIVE DISAPPEARANCE OF THE SHARP FIRST MITRAL SOUND AFTER NEOSTROPHINGIC MOBILIZATION (AUSCULTATORY DETERMINATION)

	NUMBER OF CASES	DISAPPEARANCE OF SHARP FIRST SOUND	
Complete or near complete restoration of valvular function	119	55	(46%)
Incomplete restoration of valvular function	44	23	(50%)
Total	163	78	(42%)

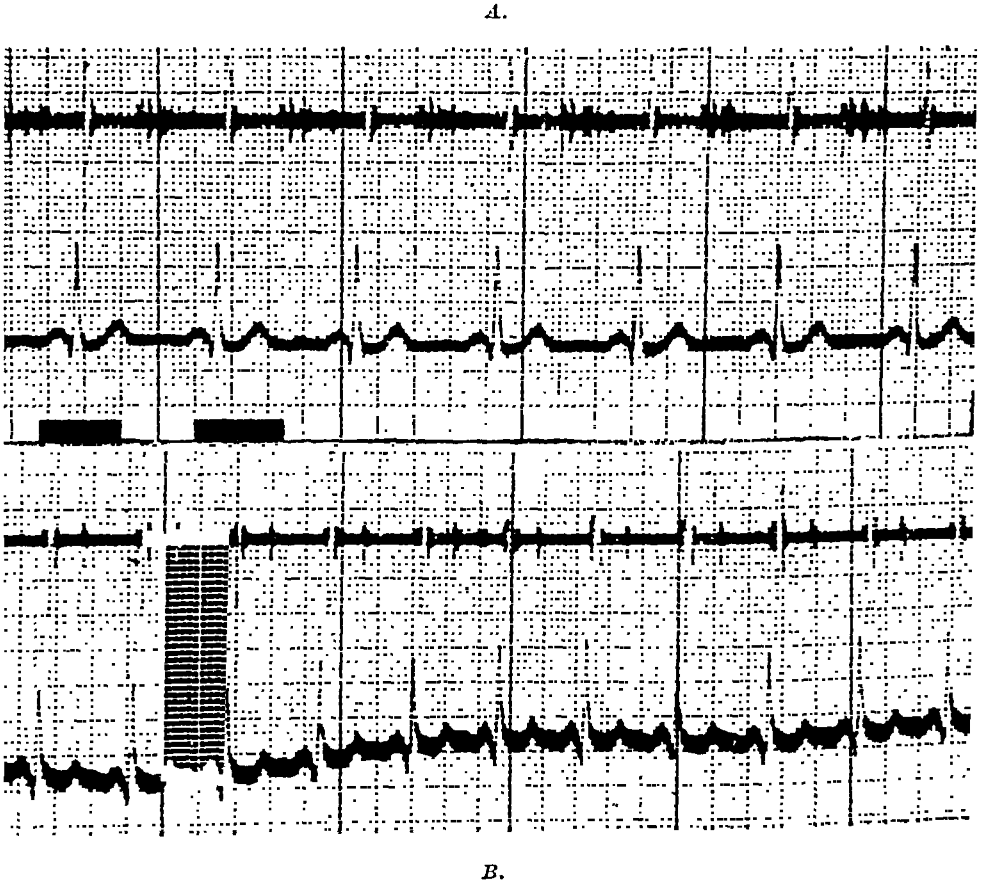


Fig. 8.—*A*, Phonocardiographic record of heart sounds at apex prior to neostrophingic mobilization of stenotic mitral valve in Patient C. A. (Preoperative.)

B. Postoperative (3 weeks) phonocardiographic study in Patient C. A. showing complete abolition of the diastolic murmur.

A complete and thorough examination of the left atrium should be carried out in all cases in order to rule out the possibility of a coexisting septal defect, anomalous pulmonary venous drainage, or the presence of a thrombus or intracardiac tumor before the definitive steps of the valvular surgery are undertaken.

Intra-atrial Thrombus.—Intra-atrial thrombosis was found in 10 per cent of this series. If the thrombus is securely adherent to the atrial wall, it should be left undisturbed, extraordinary gentleness being exhibited during the entire course of cardiac manipulation. In such cases, it is our custom to start anticoagulant therapy (first with heparin and later with Dicumarol), beginning 12 hours postoperatively, and continuing for about 2 weeks in order to lessen the likelihood of propagation of the thrombus. If the clot is loosely attached to the atrial wall or floating freely, it may be removed by utilizing one of the Litwak suction tips which may be passed into the atrial chamber along the palmar surface of the intracardiac finger (Figs. 10A and

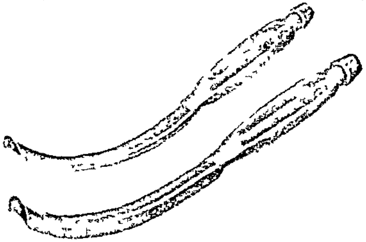


Fig 10A.—Litwak suction tips

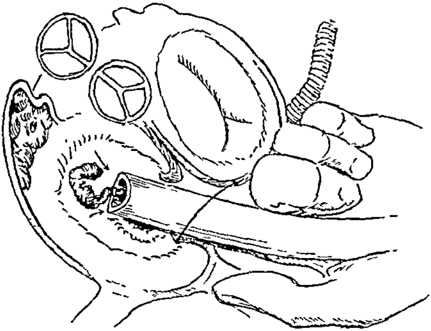


Fig 10B.—Method of aspirating floating or very loosely attached thrombotic material from left atrium by passage of suction tip into heart along the palmar surface of the intracardiac finger.

In order to prevent unexpected separation of the interatrial septum from taking place during the left atrial exploration, a 3-0 cotton stay suture is placed at either extremity of dissection to "hold" the interatrial septum together. The ends of these sutures are kept long for usage at the time of closure of the cardiac wound.

A suture of No. 2 braided nylon swedged upon a large curved needle is passed through both lips deeply beneath the line of dissection, starting at the right atrium. It traverses the septum to emerge from the left atrium and is returned at a distance of 1 cm. to form a large mattress suture. Both ends of this mattress suture are drawn upward and are loosely attached to the drapes in order to prevent subsequent entanglement. At the conclusion of the valvular surgery, this suture will be tied down to evert (Fig. 9) the lips of the incision in the left atrial wall. A pair of Morse[*] scissors (with points and outer edges of blades sharpened) is laid closed against the palmar surface of the bare left index finger which has been washed with saline and dipped into a dilute heparin solution (1:1000). The finger and scissors are inserted into the dissected portion of the interatrial groove and the scissors are advanced to puncture the exposed septal aspect of the left atrial wall. Then the blades are fully opened to produce an adequate entrance, but one which readily can be tamponaded by insertion of the index finger. As the blades are withdrawn in the partially opened position, a small gush of arterial blood will escape before the tip of the advancing index finger occludes the opening completely. The finger enters the atrial chamber slowly with a gentle twisting motion, the opening gradually becoming dilated to fit its increasing circumference.

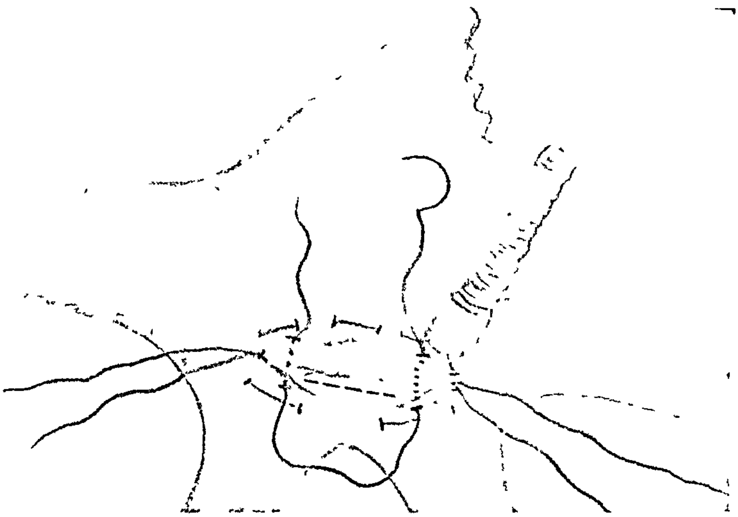


Fig. 9.—Dissection of interatrial groove and placement of various controlling sutures. Dotted line shows site of proposed incision into left atrium.

Exploration of the Left Atrium.—The finger tip soon reaches the mitral valve orifice. If the left atrium is enlarged considerably, it may be necessary to invaginate the atrial wall partially in order to reach the valve.

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[*] Available from G. P. Pilling and Sons, Philadelphia, Pa.

In most cases, persistence and repetition in application of this finger pressure soon will develop a long upward "horn" at the extremity of the horizontal portion of the valve slit. The tip of this "horn" will come to run almost parallel with the annulus fibrosus, usually at a distance of 3 to 5 mm. from it, but sometimes practically along its margin. Obviously, this latter type of cleavage is the result of deformation and retraction of the valvular ribbon since the orifice normally does not approach the annulus. Careful examination of the apex of the split after each stroke of the finger will reveal even the slightest new increment of regurgitation. This will be felt as a localized high velocity systolic jet if small, or more diffuse, involving a greater portion of the aperture, if it is larger. Should such a jet appear at the extremity of the valve slit, it is probable that the splitting has deviated from the proper line of cleavage. No further attempt at separation of that pole of the valve is then permissible lest it lead to the creation of a serious or even lethal amount of insufficiency.

After commissural separation has been completed at one pole of the valve, at attempt should be made to split the opposite one (Fig. 11, *C*).

Instrumental Separation of Fused Valve Leaflets.—Although digital mobilization of the valve leaflets is more efficient and feasible by the right-sided route than from the left, many valves still will require definitive instrumentation. For the right-sided approach, it has been necessary to modify our original right and left guillotine knives, both with respect to length and to the arc of curvature.[26, 27] Further, the principle of the Nichols papillotome[24] has been incorporated into the design.*

The anterior lip of the dissected interatrial groove is picked up with a pair of Nelson forceps and the appropriate closed guillotine knife is passed into the left atrium along the palmar surface of the operator's left index finger.

It is our policy first to incise the more favorable valve extremity (unless it already has responded to digital pressure). Often this will be the anterolateral (really left). The left-sided guillotine knife is passed through the valve opening and is brought to bear against the apex of the left extremity of the nearly transversely directed valve slit. As the jaw is opened by retracting the sliding blade, the under-beak will slide beneath the valve margin.

Digital palpation of the chordopapillary supports now is indicated to establish whether the under-beak of the instrument has been placed properly between the two parallel rows of supporting chordae or possibly through the mid-portion of a chordopapillary fusion mass. It may be necessary to rotate the instrument slightly, taking care that it does not slip from its position in order to ensure a horizontal incision at this valve extremity. By compressing the handles, the upper blade of the instrument is caused to cross the lower, shearing through 4 mm. of scarred or calcified marginal valve tissue. The closed instrument is withdrawn slightly while the finger tip remains poised momentarily over the site of section to detect even the slightest change in valve competence.

Usually it is wise to wait a few moments for reassurance from the anesthetist that the patient's condition has remained at, or has been restored to, a satisfactory level.

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*Obtainable from Storz Instrument Co., St. Louis, Mo.

Examination of the Valve.—The finger tip is held poised over the valve orifice at a distance of 1 cm. in order to detect a possible regurgitant jet. If noted, its exact localization is determined and an estimate is made of its severity. In carefully selected cases such as in this series, such a jet should never exceed a one or two plus magnitude and nearly always will be the former. Usually it will be felt to be located at the central portion of the slit-like opening.

The atrial aspect of the valve is palpated to determine the size and configuration of the mitral orifice and to recognize the possible presence of calcification.

In most instances, the valve slit will not admit a finger tip and will be centrally located with respect to the annulus fibrosus which is readily discernible from this approach.

If calcification is present, its type and amount, and especially any encoachment toward the annulus fibrosus, should be determined carefully.

Before attacking the valve, the operating table should be tilted in order to elevate the left side of the patient by 20 to 30 degrees. In this posture, the alignment of the extremities of the residual valve slit will be fitted nicely for instrumentation by either the right or left valvulotome, should such be needed. However, it is always our practice first to attempt to separate the valve structures by digital pressure.

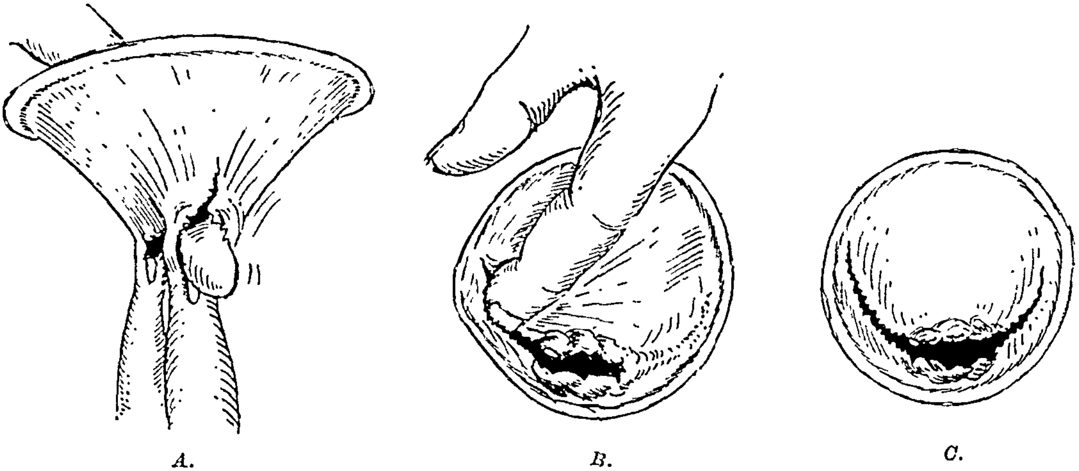

Fig. 11.—*A*, The splitting of the diminutive valve slit may be started by simply forcing the finger through the opening, or (cautiously) by application of lateral pressure.

B, The split is extended by lateral or "paper-cutter" digital pressure until the septal mobilization is complete at this pole.

C, An attempt is made to develop a similar extensive line of separation of the opposite valve pole.

MOBILIZATION OF THE VALVE

Digital Separation of Fused Valve Leaflets.—The intracardiac finger is passed forcibly through the diminutive valve slit. In favorable cases, the fused valve leaflets will begin to separate at one or both commissures. Should this occur, an attempt will be made to increase this by intermittent passage of the finger over the apex of the slit in the direction of the ventricular apex in a "paper-knife" fashion. Direct lateral or end-on pressure against the apex of the split may also be tried, always cautiously, however (Fig. 11, *A* and *B*).

will turn to the posteromedial (really right) pole of the valve This time the right sided guillotine knife will be used in much the same manner described for the left sided one

However, it is slightly more difficult to locate the proper line of separation anatomically or functionally since the extremity of the valve may be rounded rather than angular, curves less steeply upward than the anterolateral one Again, digital localization of the subvalvular supporting structures will guide the surgeon

The same precautions should be taken not to deviate from the proper line of separation (which again is more functional than anatomic) (Fig 12, *A*, *B*, *C*, *D*)

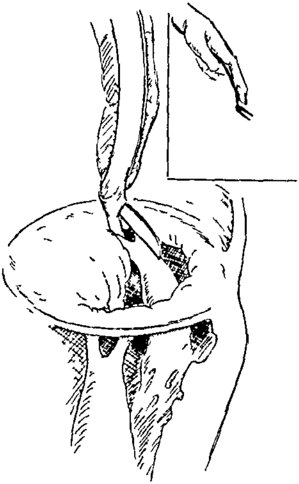

Fig 13 A —The longitudinal separation may be started in difficult cases especially those with a fibrosed short yoke by dividing the crotch with a specially designed pair of scissors

The Treatment of Subvalvular Changes—In every case after valve separation is deemed to have been obtained as fully as feasible, a search should be made for an element of subvalvular obstruction If such is found, removal of this obstruction is essential to complete renovation of the valvular mechanism

An attempt should be made to separate every abnormal chordopapillary fusion whether the papillary muscle is directly adherent to the valve apex or whether a "short yoke" chordopapillary mechanism is involved Usually, direct pressure with the finger tip against the top of the mass will suffice to start a longitudinal splitting of the muscle mass which may and should be extended fully to the ventricular attachment If the atrial extremity of this papillary mass is unusually resistant, it can be divided by a pair of special papillary

Now, an attempt should be made to split the partially divided commissure with the intracardiac finger tip. If this succeeds, no further instrumentation will be necessary and the guillotine may be removed from the heart. However, it may be found necessary to use the knife one or more additional times. In such a situation, the valvulotome should be readvanced and re-engaged at the new valve "apex." This time, the knife must be angulated upward at 30 degrees from the horizontal line and the new bite be taken in this direction.

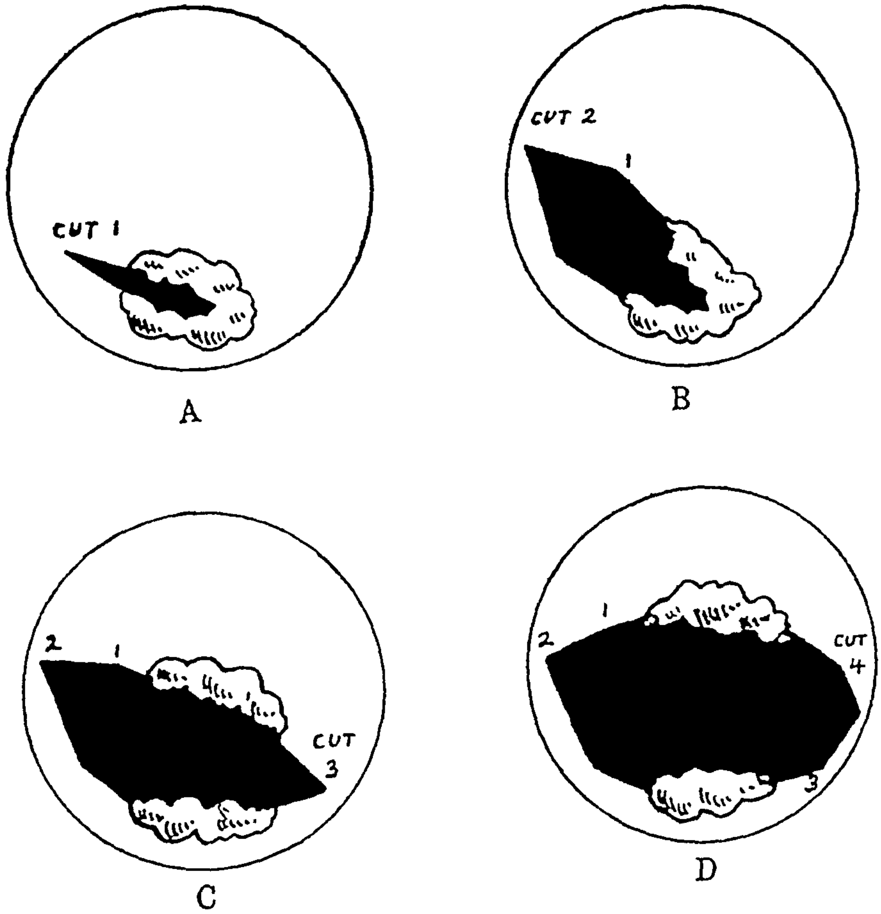

Fig. 12.—*A*, Diagrammatic representation of nearly horizontal section accomplished by first bite of guillotine against the anterolateral (really left) commissure.

B, Second (and perhaps even a later) bite is directed at an increasing upward angle in order to follow the natural curve of the line of valve closure. Shaded area indicates increase in size of orifice.

C, First bite against the posteromedial (really right) commissure is directed horizontally.

D, Any additional instrumental division of this pole must be guided in an increasingly upward fashion. Note extensive increase in effective valve area (shaded portion).

If still another bite should be required, this should deviate further upward from this line (almost 60 to 90 degrees from the original line). At the tip of a well-formed horn, the line of section is nearly vertical to the mid-portion of the valve slit.

If digital pressure becomes effective at any time, the valvulotome should be withdrawn completely from the heart and the separation continued digitally.

As soon as a satisfactory (or compromised because of regurgitation) opening of the anterolateral pole of the valve is obtained, the operator's attention

Exploration of the Tricuspid Valve—Routinely, the tricuspid valve should be explored[18 26 27] by inserting the finger through an incision made into the tip of the right auricular appendage if the patient's general condition permits this step Since, as shown previously, one fourth of all the cases of mitral stenosis have a certain degree of tricuspid valvular involvement and 8 per cent require definitive surgical correction of stenosis, it usually is worth while to spend another extra ten minutes of operating time in examining this valve

OPEN HEART SURGERY FOR MITRAL STENOSIS

Obviously a similarly extensive type of valvular mobilization might be accomplished by an open technique using one of the existing pump oxygenator combinations Here, too, the use of the right sided approach is essential, but the left atrium must be incised longitudinally for a greater distance Usually the incision is made just dorsal and parallel to the interatrial groove

However, because the appropriate line of valve division is not strictly an anatomic but rather a functional one, with an open technique the operator is at a distinct disadvantage in that he is unable to appreciate the earliest stage of creation of incompetence of the nonfunctioning valve While one might think that the proper line of commissural obliteration would be clearly apparent to visual inspection, it often is most confusing to decide upon its location Because of the appreciable risk of creating inadvertently a serious grade of insufficiency which cannot become evident until full cardiac action has been restored (and therefore is essentially irremediable) the operator will tend to restrict the extent of valve mobilization to less than the logical ideal Similarly, he may be loathe to split the papillary muscles fully to the ventricular wall

For these reasons, although with a personal open surgical experience admittedly limited to but 3 patients with this disease, we have felt that the expert operator will perform a better mitral valve mobilization with the simpler and safer closed technique than with the open

SUMMARY AND CONCLUSIONS

After a 9 year clinical experience including more than 2,000 patients subjected to mitral commissurotomy through the standard left sided approach, and close to 500 operated during the past 4 years by the right sided method, a new and technically much superior type of valve mobilization has been evolved It is based upon the principle of converting the irretrievably destroyed flutter-valve action of the mitral valve to an efficient flap valve mechanism

This effect is accomplished by a rehinging or new hinging of the septal cusp not only through its flexible mid zonal tissue, but also with respect to the usually shortened papillary suspension yoke which must be split longitudinally Such a maximally corrective procedure has been described under the term "neostrophingic mobilization" of the valve It cannot routinely be accomplished except with the technical advantage of the right-sided approach

It has been shown that most patients with mitral sten have r ived but partial (as shown by the persistence of auscultator scissors* which are designed to be passed into the heart along the palmar aspect of the intracardiac finger. Thereafter, splitting usually will be accomplished easily (Figs. 13*A* and 13*B*).

Closure of the Cardiac Wound.—After full (or compromised) neostrophingic mobilization of the mitral valve has been completed, the finger is withdrawn slowly from the opening in the wall of the left atrium, tamponading the wound surface as the initially placed mattress suture is finally tightened. Usually this will establish complete hemostasis. Should it fail, tightening of the purse-string suture will supplement it. If there still is persistent bleeding, the two lips of the dissected interatrial groove may be approximated with Nelson forceps while they are being sutured.

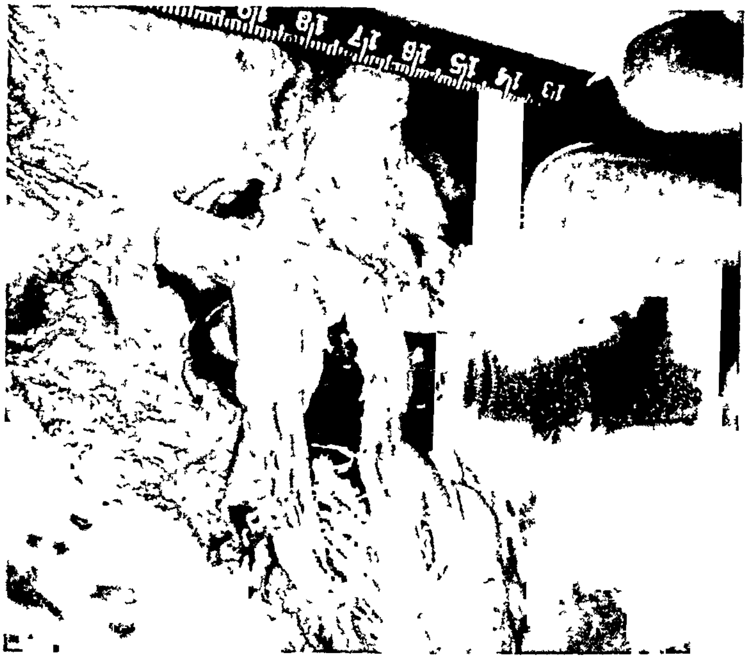

Fig. 13*B*.—Autopsy performed operation showing fully mobilized septal leaflet, both directly adherent papillary muscles having been split to their ventricular wall attachment.

The dissected groove is repaired with one continuous horizontal everting suture and one re-enforcing simple suture using the long ends of the previously placed 3-0 cotton stay sutures.

After thorough flushing with normal saline, the pericardium is closed tightly enough to prevent herniation of the heart to the right, but loosely enough to permit free drainage of any accumulated exudate into the chest cavity.

Two multiwindowed catheters are placed intercostally for postoperative drainage and are connected with a water-seal drainage bottle. The ribs are approximated with pericostal sutures of No. 2 double chromic catgut. The muscles and fascia are closed with continuous No. 1 chromic catgut sutures. Subcuticular No. 30 steel wire is used for skin closure.

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*Obtainable from the Storz Instrument Company, St. Louis, Mo.

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temporary relief of their valvular obstruction when the older left-sided commissurotomy procedure has been carried out. Restenosis ultimately may be expected in a large proportion of these patients who have, in actuality, been subjected merely to linear (and limited) division of a fibrous stricture. It is believed that recurrence of mitral stenosis after complete neostrophingic mobilization will be rare indeed since the operation amounts actually to construction of a valve mechanism of different type, and since all the tissue cleavages extend well into normal unfibrosed structures.

The early operative mortality and morbidity associated with complete reconstitution of the valve is distinctly less than that associated with less complete operations (see Tables VIIA and VIIB).

Because of certain unavoidable handicaps experienced during attempts to carry out an extensive mobilization of the valve structures with maintenance of full competence when the open technique is employed (using the pump-oxygenator), it is felt that a better and safer reconstitution of the stenotic mitral valve can be achieved by the closed method.

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including the crista supraventricularis, tricuspid valve, and the papillary muscle of the conus (Fig. 2) Through a right ventriculotomy one perceives that he is first looking into a deep recess, the medial wall of which is formed by the defective ventricular septum. The opposite or lateral wall of this recess appears to be made up, at least in part, of tricuspid-valve leaflet, whereas superiorly the crista supraventricularis narrows the opening into the recess. The opening itself may be bridged by at least one papillary muscle of the conus. At the bottom of this recess is a central fibrous body (or fibrous trigone) to which the aortic-valve ring is firmly attached on the left side, and the tricuspid-valve ring on the right. If one then looks from a caudal point and has proper lighting, he can look into the recess, thence through the ventricular septal defect, and see the aortic cusps.

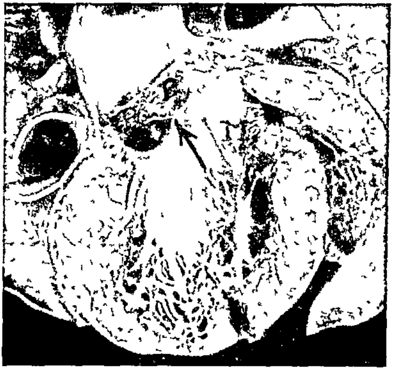

Fig 1—Specimen from a 3-month-old boy, showing the ventricular septal defect from the left ventricular aspect. The defect lies inferior to the adjacent halves of the right (*R*) and posterior (*P*) aortic leaflets The arrow points to the region of the central fibrous body, where the ventricular septum and the aortic, mitral, and tricuspid-valve rings meet It is essential that the operative repair of high ventricular septal defects begin in this area

Thus, a striking contrast exists between the appearance of this particular type of high ventricular septal defect when viewed from its right and left ventricular aspects Indeed, the simplicity of the relationships and the unobstructed view from the left might raise doubt as to the wisdom of approaching the defect through the right ventricle. The impracticability of a left ventricular approach surgically, however, has required persistence in attaining an anatomic orientation from the right, and from this beginning to develop a reproducible technique for complete closure of these defects.

SURGICAL TECHNIQUE

The first requirement for the surgical correction of ventricular septal defects is obviously a means for whole-body perfusion which can be safely maintained for as long as is necessary for the repair. Induced cardiac arrest has clearly become a fundamental necessity to the accurate repair of ventricular

SURGICAL TECHNIQUE FOR REPAIR OF HIGH VENTRICULAR SEPTAL DEFECTS

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THE time-honored requirements for accurate open operation in any region are a well-exposed, quiet, and relatively bloodless field, and a precise and reproducible operative technique. Preoccupation with the problems of whole-body perfusion must not obscure the necessity for the development of adequate techniques for the reparative intracardiac operations thereby made possible.

One of the challenging intracardiac procedures currently performed is the closure of high ventricular septal defects. These are defects located immediately beneath the aortic-valve ring, and thus high in the ventricular septum when viewed from the left ventricular aspect. Approximately 90 per cent of patients having ventricular septal defects who are selected for surgical treatment and who, therefore, have significant hemodynamic derangement, have a ventricular septal defect in this region.

In a previous publication[1] in this JOURNAL, a technique used at the Mayo Clinic for repair of these defects was described, but a footnote to the report indicated it was no longer in use in this institution. Experience with the repair of ventricular septal defect, either alone or as part of the tetralogy of Fallot, has led to gradual modifications in technique. The method herein described has been used since the adoption of purposeful cardiac arrest and we consider it to be a definitive method.

ANATOMY

Closure of this particular defect requires a clear three-dimensional concept of the compact, complex, and somewhat obscure anatomy of this region.[1, 2] The complexities of the embryologic development of this area of the ventricular septum are beyond the scope of this presentation.[3]

On considering a high ventricular septal defect, such as has been described, from the left ventricular aspect only, the anatomic relationships can be easily comprehended and expressed. The defect presents as an opening that usually equals or slightly exceeds the aortic orifice in diameter. In most instances, the defect is immediately inferior to the right aortic cusp, the adjacent portion of the posterior aortic cusp, and the intervening commissure, and there is no superior remnant of ventricular septum below the aortic ring itself (Fig. 1).

From the right ventricular aspect, however, the region of the defect is partially obscured and rendered relatively inaccessible by adjacent structures,

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As mentioned previously, it is usually beneath the right and posterior cusps and their intervening commissure, but it may be beneath one of these more than the other. The correct identification of the position of the aortic ring is basic to the planning of the repair.

It is essential to realize that maximal protection to the integrity of the repair requires that closure be accomplished without undue tension. The power of the heartbeat can disrupt a repair done either with or without a prosthesis if the repair is made in an improper direction. These defects are usually ovoid, and the line of repair must be parallel to the long axis of the oval. This line is usually parallel to the aortic ring and transverse to the outflow tract of the left ventricle (Figs. 3 and 4). It is impossible to overemphasize the importance of this proper line of repair. To accomplish repair in this proper direction, the surgeon must constantly judge the left ventricular relations of the defect while working through the right ventriculotomy opening.

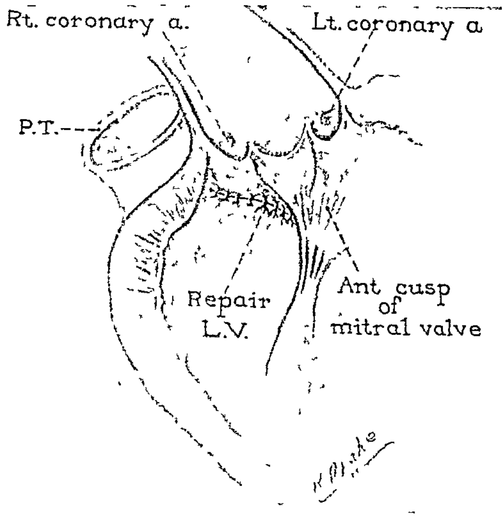

Fig 3.—Completed repair of a high ventricular septal defect by the direct-suture technique, as seen from the left ventricular aspect Note the relation of the direction of the resultant suture line to the aortic-valve ring.

As the defect is viewed from the left ventricular aspect, its upper margin consists essentially of aortic ring and its lower margin of ventricular septum. When viewed through a right ventriculotomy incision, the previously described upper margin lies superiorly and to the patient's right, and the lower margin lies inferiorly and to the patient's left. When the ventricular septum is viewed from the left ventricular aspect, the central fibrous body lies in the region of the extreme posterior corner of the defect, or to the viewer's right. When septal defects. The surgical approach is through right ventriculotomy, usually extending from a point just below the pulmonic-valve ring downward nearly to the margin of the heart.

Small cat's-paw retractors are placed at three points about the edges of the ventriculotomy opening, not only to hold the wound open but also to support the heart. Variable degrees of tension on each of these retractors will give variations in the exposure of individual intracardiac regions. The intracardiac sucker can be placed through the tricuspid valve into the right atrium and steadied in such a manner that it not only keeps the field bloodless but also assists in exposure. Other retractors inside the heart are not usually utilized, but movable structures, particularly chordae tendineae, may be manipulated by gently grasping them with fine tissue forceps.

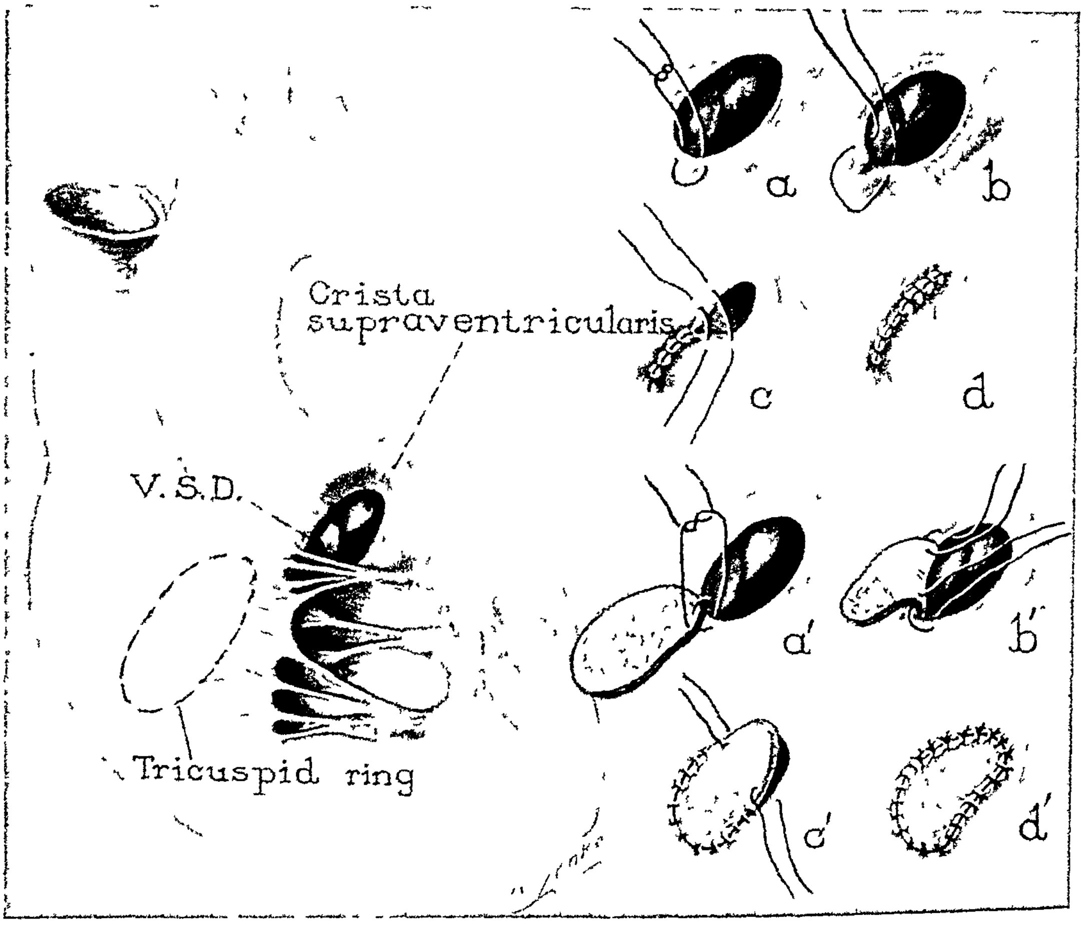

Fig. 2—Schematic representation of a high ventricular septal defect as viewed from the right ventricular aspect. The insets demonstrate the steps in the surgical repair by both direct suture and insertion of a prosthesis, as described in the text. Note that certain stitches were placed from the left ventricular side, working through the defect, and that the knots come to lie on the left side.

Once exposure has been obtained through the right ventriculotomy, the surgeon must orient the defect with relation to the *left* ventricular structures, since these are basic to the planning of the repair. First, the defect is identified as a high one by noting that it is immediately beneath the aortic cusps ventricle, and then passing the needle from left to right through the ventricular septum at its junction with the central fibrous body and tricuspid ring. To complete this first stitch the needle is passed from right to left through the corresponding fibrous tissue superiorly at the junction of aortic-valve ring with tricuspid-valve ring. This suture thus grasps tissue at each extremity of the central fibrous body, and is tied with just sufficient tension to approximate the tissues snugly. This is the key suture—the principal one that assures a complete repair (Fig. 2, *a*).

Further interrupted sutures are then similarly placed at intervals of 2 to 3 mm Each of these is first passed through the superior margin of the ventricular septum (or the lower margin of the defect) from the left ventricular aspect toward the right. Superiorly, each suture passes through the surprisingly tough tissue of the free margin of the aortic-valve ring, care being taken not to stitch into aortic-valve cusp (Fig. 2, *b*). Working in this fashion, each suture successively placed for repair of the defect lies more anteriorly than the previous one.

Because of the fact that each of the sutures placed thus far passes from the left ventricle to the right and then back again, the knots come to lie on the left ventricular aspect. This has not resulted in discernible undesirable sequelae

After about half of the defect has been closed in the manner described, it will be found that the aortic ring gradually fuses with the deep surface of the muscle of the crista supraventricularis and disappears behind that structure. The crista itself at this point appears to form the upper margin of the defect, and there is no contraindication to regarding it as such. Therefore, a change is made in the method of suturing. The stitches now are made entirely from the right ventricular aspect, passing through the lower edge of the crista supraventricularis above and through the ventricular septal margin below, with the knots now lying on the right ventricular surface of the septum (Fig. 2 *c*). As these are placed, the remainder of the repair is progressively completed (Figs 2, *d*, 3 and 4). As the final sutures are placed, it is important that the left ventricle be filled with blood and that residual air be expressed, it sometimes being necessary to inject blood into the left ventricle through the defect before the last sutures are tied.

Repair by Inserting a Prosthesis.—If the choice is made to utilize a prosthesis in the repair, the basic method is similar to that for direct repair. The prosthesis is first fashioned from a piece of polyvinyl sponge compressed to 3 mm. in thickness,* oval in shape, and only slightly smaller than the defect itself. The first suture is placed precisely at the same point as for direct repair, except that here, of course, each suture takes only one bite into tissue, passes through the polyvinyl sponge and then is tied. As this first suture, placed in the region of the central fibrous body, is tied, the prosthesis is lowered into position It is helpful to conceive of this first suture, when tied, to be functioning as a hinge upon which the polyvinyl sponge can swing just as a viewed from the right ventricular aspect, it is situated far posteriorly and beneath the aortic ring in a relatively inaccessible angle. This inaccessible angle is of critical importance in obtaining a complete and secure repair. It is essential that the repair begin in this region, for it is quite impossible to expose the posterior aspect of the defect properly and hence to accomplish complete repair if the suturing is begun at its opposite or anterior end. Furthermore, by beginning at this angle, this portion of the repair can actually be performed from the left ventricular side by working through the septal defect, thus obtaining not only additional space but also improved orientation which a left-sided approach provides.

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*Compressed to approximately a fourth of its original thickne

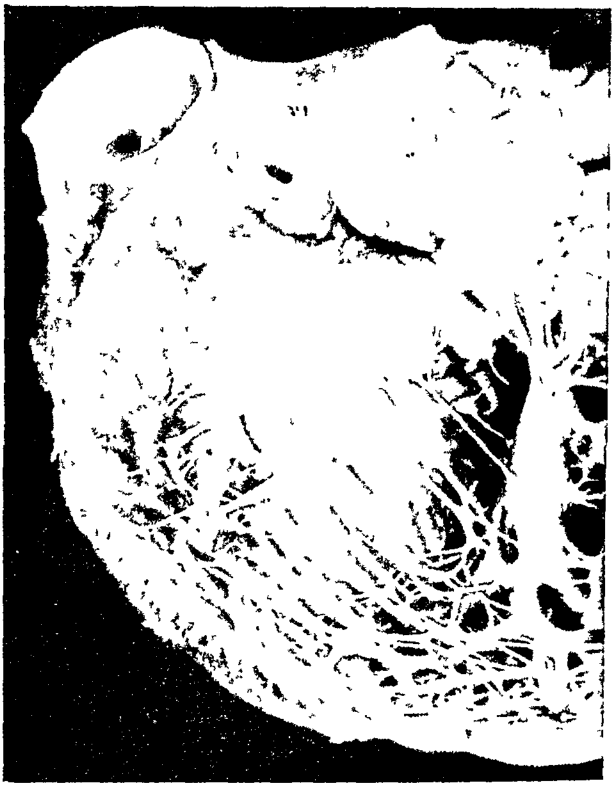

Fig. 4.—Necropsy specimen of a high ventricular septal defect repaired at operation by the direct-suture technique, as viewed from the left ventricular aspect. The suture line is partially obscured by the overhanging aortic valve cusps. The patient died 48 hours after operation as the result of an associated severe congenital deformity of the mitral valve which could be only partially corrected. The defect was a very large one, but the repair is obviously without tension and structures are not distorted.

Only after study of the defect and its relations is completed should suturing begin. Number 3-0 silk threaded into a small sturdy needle (Adson dural needle) is employed. Direct suture is at present the technique chosen to repair nearly all of these high defects, including those which are very large. Since it is not certain that use of a prosthesis is to be completely abandoned, this technique of repair also is described.

Direct Suture.—The first stitch in a direct repair is begun by placing the tip of the needle holder through the ventricular septal defect into the left

COARCTATION OF THE AORTA WITH A REPORT OF AN UNUSUAL OPERATIVE COMPLICATION

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THE majority of patients with coarctation of the aorta may be treated surgically with a good chance of obtaining a satisfactory result Inasmuch as the average life expectancy of the untreated case is approximately 30 years, it behooves all physicians to endeavor to discover the patients with this defect early so that surgery can be performed before irreversible changes have occurred The infantile type poses a more serious problem, since there is a long coarctate segment and, in addition, there are often associated cardiovascular defects The surgical mortality rate is highest in the patients operated upon in infancy or early childhood and after late adolescence, or early adult life When surgery is delayed until after age 30, a more serious appraisal should be made before the decision to proceed is made The ideal age for surgical correction is perhaps in late childhood when the aorta has attained a substantial size and before serious degenerative changes have taken place The over all operative mortality rate at the present time is somewhat less than 10 per cent and may be expected to decrease significantly as earlier diagnosis and surgery is achieved

The method of treatment of coarctation is entirely dependent upon the length of the narrowed segment, the availability of grafts, the position of the involved area, and the degree of associated cardiovascular defects of a congenital nature The method proposed by Blalock and Park[1] involves an anastomosis between the left subclavian artery and the distal aorta Crafoord and Gross,[2, 4] in 1944, each operated upon patients by resecting the coarctation and effecting an end to end anastomosis of the aorta This method has continued to be the most effective method of surgical correction of coarctation and is used whenever possible With the advent of common use of both homografts and synthetic plastic grafts, longer defects can now be bridged without great difficulty

The majority of operative complications which result in death are those due to the direct effect upon the heart at the time of surgery These include cardiac arrest, ventricular fibrillation, irreversible shock, and heart failure

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***Assistant Professor of Surgery University of Neb Med [illegible]ool trap door would swing on its hinge. Initially, this door is held open toward the right and against the tricuspid valve, thus leaving the ventricular septal defect open and permitting the placement of further sutures from the left ventricular aspect (Fig. 2, *a′*). Further simple interrupted sutures are thus placed both superiorly and inferiorly about the deep margins of the defect (Fig. 2, *b′*). Again, as the aortic ring is followed superiorly and anteriorly, it fuses with the musculature of the crista supraventricularis, and at this point the unattached portion of the prosthesis is swung to the left, completely closing the defect and permitting the remaining sutures to be placed and tied from the right ventricular aspect (Fig. 2, *c′*).

SUMMARY

High ventricular septal defects with little or no septal remnant beneath the aortic ring present technical difficulties in their complete repair. An accurate and reproducible technique is described whereby the complete correction of such defects can be accomplished.

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to exude in large volume and consequently the pericardium was extensively incised and explored. A long rent, approximately 4 cm. in length, was identified on the right posterolateral aspect of the ascending aorta with its lowermost extension about 2 cm. superior to the aortic valve. The position of the rent made control of the hemorrhage most difficult. A running suture of No. 1 silk was used to close the defect and, following this, several points of leakage were reinforced with No. 4 0 arterial silk. There was moderate amount of ooze from the sutured defect which gradually ceased upon pressure over the area. Immediately after incision of the pericardium and release of the acute cardiac tamponade the heart regained its normal rhythm and this continued throughout the period of observation. Upon closure of the rupture and coincident with rapid transfusion of 2,500 c.c. of whole blood, the blood pressure again returned to normal where it remained throughout the completion of the procedure. In spite of the grave situation which existed, it was felt that resection of the coarctation would lessen considerably the pressure upon the sutured aortic defect. Consequently, the subclavian artery and aortic arch were mobilized, the ligamentum arteriosum ligated, and the narrowed segment was excised. An end-to-end approximation was accomplished with posterior and anterior everting, running mattress sutures, reinforced at various points with interrupted mattress sutures. The clamps were slowly released following suture and the patient's condition remained satisfactory throughout the remainder of the closure. The patient received a total of 16 units of blood (8,000 c.c.) during and immediately after operation. Examination of the specimen revealed the aorta to be totally occluded (Fig. 2). Postoperatively, the course was characterized by moderate hyperpyrexia, blood pressure fluctuations, and tachycardia. In addition, there was an obvious cl [illegible] o [illegible] sensoriu

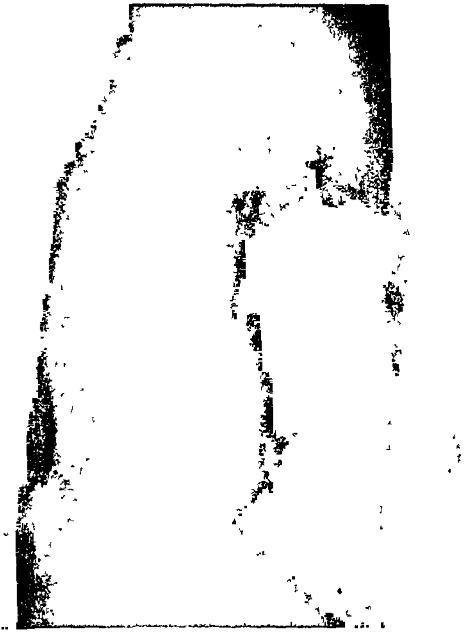

Fig. 1.—Angiocardiogram showing complete coarctation and large left subclavian artery.

Second in importance as a cause of death is disruption of the anastomosis, and third is operative hemorrhage which is usually initiated by trauma to the thin, friable, dilated intercostal vessels.

There is a miscellaneous category of operative complications which comprise a significant percentage, but are too varied in their nature to be covered in this discussion.[5-8] Among these complications is the entity known as necrotizing arteritis as described by Ring and Lewis.[9]

The case which is reported herein represents an example of a complication previously unlisted in the surgical literature to the best of the authors' knowledge. During surgery, the patient developed an extensive rupture of the proximal aortic arch with survival and satisfactory end-result.

CASE REPORT

R. C. W., a 21-year-old white man, was transferred to this hospital with a diagnosis of coarctation of the aorta. Several months prior to admission and just prior to discharge from the army, a physical examination revealed a blood pressure of 140/110 mm. Hg in both upper extremities. Presumably the patient was normotensive on previous examinations during and prior to military service. The patient had no significant complaints other than occasional numbness or a tingling sensation in the lower extremities for approximately two months. He stated that his feet had always been cold but he denied any weakness and had been active in athletics during his preparatory school days. On physical examination, the pressure in the right arm was 150/105 mm. Hg and that of the left arm 145/105 mm. Hg. Pressures in the legs were unobtainable. The body was muscular and well proportioned with no evidence of lower extremity muscular atrophy. The peripheral pulses of the lower extremities were not obtainable. The neck veins were not distended. There was no ankle edema. The chest was clear. There was no significant cardiomegaly but a harsh Grade 3 systolic murmur was noted over the aortic area. The skin of the lower extremities was damp, moist, and cold.

Laboratory data: Chest x-ray revealed enlargement of the cardiac silhouette. There was some rotation of the heart with recession of the aortic knob. There was minimal notching of the ribs in the midthorax. An esophagram revealed no fluoroscopic evidence of displacement or other significant abnormality. An electrocardiogram was considered to be within normal limits.

Shortly after admission, an angiocardiogram was performed by rapidly injecting 50 c.c. of 90 per cent Hypaque through a No. 14 polythene catheter with its tip in the axillary vein. The film taken 7 seconds after this injection shows a typical segment of coarctation immediately beyond the take-off of the left subclavian artery (Fig. 1). Several large collateral vessels were also visualized.

Following adequate preoperative preparation, the patient was taken to surgery and in the right lateral position a left infrascapular incision was made. The dissection proceeded down to the pleural cavity which was entered without difficulty. Numerous large vessels were encountered during this procedure, all of which were securely ligated. On inspection of the great vessels, the arch of the aorta was found to be somewhat smaller than normal and the left subclavian artery was enlarged to approximately the size of the aortic arch. There was a short complete coarctation 3 cm. below the take-off of the left subclavian artery. The third intercostal arteries were small, but the fourth intercostal arteries and the internal mammary arteries were markedly dilated. The mediastinal pleura was incised over the aorta extending upward between the vagus nerves and left subclavian artery. Mobilization of the aorta and the left subclavian artery was being accomplished when a cardiac arrest was noted.

The lung was retracted from the heart surface and cardiac massage was immediately begun. However, it was noted that pressure against the myocardium was very resistant. In order to secure more effective massage, it was decided to incise the pericardium and upon so doing a large quantity of blood gushed from the pericardial cavity. Blood continued

SUMMARY

A brief discussion of the necessity for surgical treatment for coarctation of the aorta is given. A most unusual and previously unreported complication occurred at operation, namely, rupture of the proximal aortic arch. This was successfully sutured and a satisfactory result obtained.

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However, one week following the operative procedure, the patient appeared in all respects normal with the exception of episodes of tachycardia. The patient's memory returned without defect and his intelligence seems in no way disturbed. Tests by psychologists rate him as a high normal. The use of heavy dosage of antibiotics was continued for 3 weeks following surgery. After only minimal difficulty with postoperative hypotension the blood pressure has stabilized at about 105/70 mm. Hg, and has remained at this level for 3 months postoperatively.

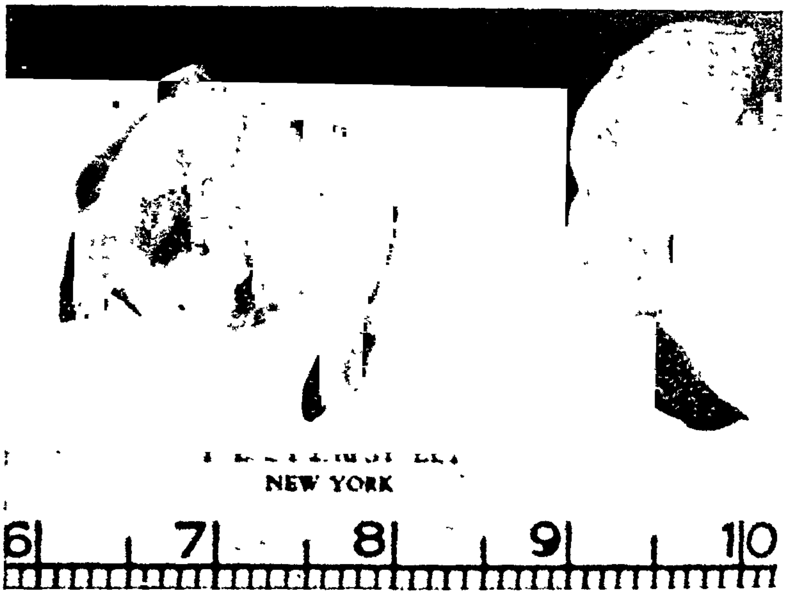

Fig. 2.—Excised coarctate segment.

DISCUSSION

It has been reported by Reifenstein and co-workers[10] that, of 104 autopsied cases of adult type of coarctation, approximately one fourth of patients died from rupture of the aorta. An additional one fourth succumbed from endarteritis or endocarditis, one fourth from cerebral vascular accidents, and one fourth from causes not related to the cardiovascular system. Upon reconsideration of the events immediately prior to operation, it was recalled that the patient complained of mild to moderate substernal pain somewhat to the left of the mid-sternal line. This had been noted on several occasions for approximately one week prior to operation, and it was felt by the authors that this represented apprehension which the patient was experiencing as the date of his surgery approached. In retrospect, it appears that the pain indicated the first events of what was to be a rupture of the aorta. Due to slight blood pressure increase during the minimal trauma associated with mobilization of the aortic arch, the rupture did occur and fortunately was quickly identified and repaired. The anesthetist reported no significant fluctuation in blood pressure prior to the cardiac arrest, although, presumably this could have been momentary and not noted during the routine blood pressure determination. Although further examination during the procedure was limited because of the gravity of the situation, there were no other outstanding cardiovascular defects noted.

The distal part of the autograft was then sutured to the left main bronchus. Necrosis occurred in that autograft. In another dog, a left pneumonectomy was performed first and the left main bronchus was then used as an autograft replacing 8 rings of the trachea (Fig. 2). In this case, first a left thoracotomy was performed for the pneumonectomy, then a right thoracotomy for the tracheal replacement. Necrosis occurred, however, in the autograft (Fig. 3, *A* and *B*). In the third dog, first a left thoracotomy was performed with extirpation of the left upper lobe and with a direct anastomosis of the left upper lobe bronchus to the left lower lobe bronchus. Thus, the lower lobe remained in the left thorax. The main bronchus was divided at the level of carina. Then a right thoracotomy was performed, the trachea resected, and the left main bronchus used for tracheal reconstruction. The divided bronchus to the left lower lobe was then anastomosed to the right lung. Thus, a complete reconstruction of the tracheal bifurcation was obtained. A necrosis occurred, however. As necrosis occurred in a main bronchus without connection to its lung when it was used for reconstruction of the trachea, this necrosis was thougl due to lack of blood supply through that pulmonary artery and the ts w changed in the following way.

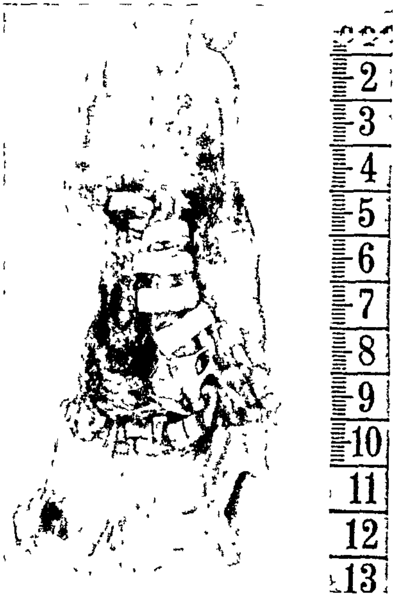

Fig 1.—Specimen from dog, in which a freeze-dried tracheal homograft was inserted after resection of 10 rings of cartilage of the trachea. After 15 days, necrosis of the mucosa occurred and the cartilage rings were lying loose inside the lumen of the trachea, causing obstruction and stenosis breathing Fifteen days after the insertion, the other specimens had a similar appearance

RECONSTRUCTION OF THE TRACHEA AND ITS BIFURCATION

An Experimental Study

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Large tracheal defects necessitating circumferential reconstruction present a difficult problem, especially when the carina is involved. They must be repaired with some material which is airtight and semirigid and with a minimum of tissue reaction. Daniel[6] has shown that tubes of Vitallium, stainless steel, and glass will provide a temporary airway but will not give a good long-term result. Rob and Bateman,[9] Bucher and co-workers,[7] Edgerton,[10] and Keshishian and co-workers[5] have used stainless steel and tantalum mesh, while Belsey[8] used a wire coil wrapped with host tissue for tracheal replacement. Excellent experimental results have been obtained by Ekeström,[2] who embedded subcutaneously a plastic frame, placed over a plastic tube wrapped in fascia, in the experimental animal for 3 to 12 weeks prior to the reconstruction. At the reconstruction, the inner tube was removed when an autologous connective tissue tube, reinforced with a plastic (Teflon) frame, was obtained.

EXPERIMENTAL RESULTS

1. *Homografts.*—Preserved homografts have been used earlier by Keshishian, Blades, and Beattie.[5] These tracheal grafts were preserved in modified Tyrode's solution with antibiotics and maintained at -4° C. No grafts were older than 14 days. These homografts seemed to be well healed after one week but, in the period of 3 weeks, there was a beginning liquefaction and necrosis, leading to stenosis and death. We have tried to desensitize homologous transplants of trachea, in the following manner.

Segments containing 10 to 11 rings of cartilage of the trachea were freeze dried and then implanted in the trachea in 4 dogs. All dogs died due to degenerative changes of the homografts. The rings of cartilage rolled into the lumen of the trachea, causing stenosis and death (Fig. 1). In 2 dogs, a formalin-fixed tracheal homograft, either 40 or 4 per cent formalin, was used with the same disappointing result. In another 2 dogs, a tracheal homograft preserved in 95 per cent alcohol was used, with liquefaction and necrosis developing in 2 to 4 weeks.

2. *Autografts.*—In a 15 Kg. dog, a piece of trachea containing 13 rings was resected and then a right pneumonectomy was performed. The right main bronchus was then used for the reconstruction of trachea in the following manner. The autograft was first sutured to the transected end of the trachea.

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3. *Direct Anastomosis.*—Through a right thoracotomy, a wide dissection was made of the trachea, the carina, and both main bronchi. After resection of 12 tracheal rings, the right main bronchus was sutured end-to-end to the trachea Then the left main bronchus was sutured end-to-side to the intermediate bronchus below the origin of the right upper lobe (Fig 4). Thus, both lungs were preserved yet the lower part of trachea with the carina could be resected. This proved to be a rather difficult procedure in a small dog as the quality of the left main bronchus was not good enough for anastomosis end-to-side to the right intermediate bronchus. The cartilages of the left main bronchus were too soft and too small The experiment was therefore repeated in a 30 Kg dog which made an uneventful recovery for 3 months and then died due to infection and atelectasis. There was an abscess around the suture line to the left lung with fibrosis and degeneration in the main bronchi This, however, seems to be the most promising method in the resection of the carina with the distal part of the trachea, i e., to do an end-to-end anastomosis between the right main bronchus and lower end of the trachea and then to implant the left main bronchus in the right main bronchus. Due to the good results obtained in 12 clinical cases of bronchotracheal anastomosis, this method of direct anastomosis for carinal reconstruction is considered the best method available. It will, however, be difficult to perform this operation in dogs due to the soft cartilage of the left main bronchus. A similar method has already been successfully applied to a clinical case by Mr. R. Barclay.

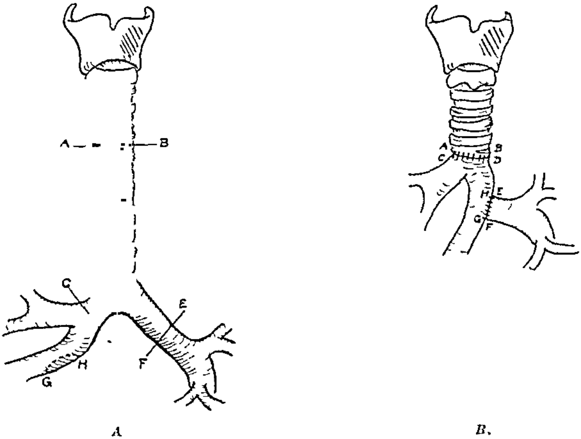
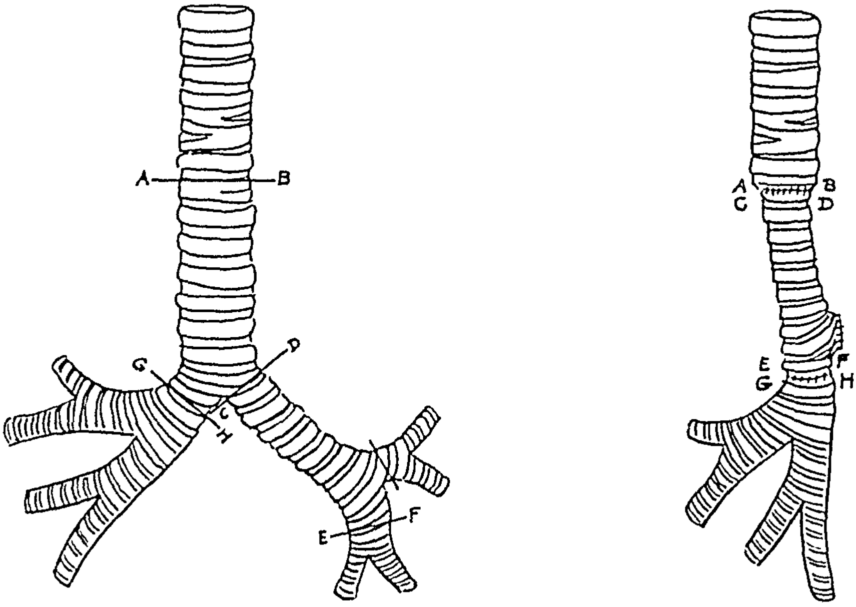


Fig 4.—*A*, The trachea, carina, and first parts of the left and right main bronchi were resected Then an end to-end anastomosis was made between the distal part of the trachea and the right main bronchus Then, the left main bronchus was anastomosed end-to-side to the intermediate portion of the right main bronchus

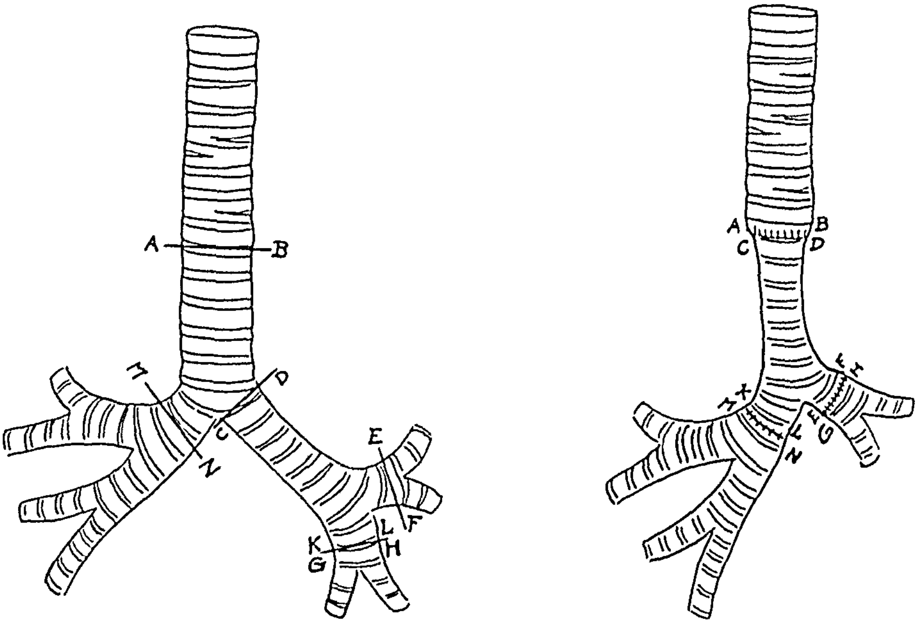
B, An anastomosis completed after resection of the trachea and carina with preservation of both lungs



A. *B.*

Fig. 2.—*A*, The left main bronchus was used as an autograft for replacement of trachea. First, the left thoracotomy with left pneumonectomy was performed. The left main bronchus was then, by means of a right thoracotomy, used for replacement of the trachea.

B, The left main bronchus has been inserted in place, connecting the distal end of trachea to the right main bronchus.



A. *B.*

Fig. 3.—A, First, a left thoracotomy was *performed with resection of* the left upper lobe. The left upper lobe bronchus was then anastomosed to the left lower lobe bronchus. *The left main bronchus* was *divided at the level of the carina and the* left thoracotomy wound was closed.

B, A right thoracotomy was then performed. The left main bronchus was drawn up in the wound and anastomosed to the distal cut end of the trachea. The original left lower lobe bronchus was then anastomosed to the right main bronchus. Thus, a complete reconstruction of trachea and carina, with first parts of both main bronchi, was obtained. Necrosis occurred in the autograft.

Juvenelle has already shown, in 1951, that it is possible to remove the lowermost part of trachea with its bifurcation. But with a resuture of both main bronchi end-to-side to the trachea of a dog, it is not possible to mobilize the left main bronchus for any significant distance It is, however, possible to remove a longer section of trachea when an end-to-end anastomosis between right main bronchus and the trachea is performed and the left main bronchus is sutured into the right main bronchus, end-to-side. The suture line must not be under too much tension, otherwise it may cause a stricture to form. From these dog experiments, it seems that there is a more pronounced tendency for stricture formation when an end-to-end anastomosis of a main bronchus to the trachea is made under tension as compared to a bronchotracheal anastomosis.

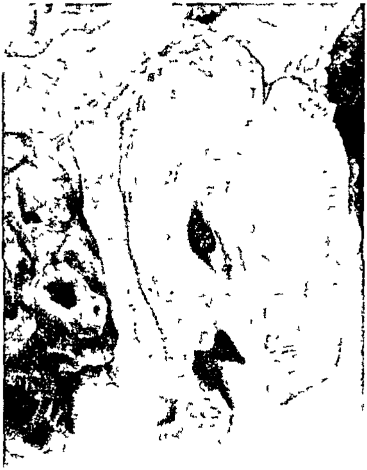


Fig 7 —Perforation of the abscess, around a prosthesis, into the esophagus In another case, there was erosion of the carotid artery.

In 1950, Ferguson and associates[1] reported a study of primary tracheal anastomosis They concluded that when stenosis, following repair, did not exceed one third of the original lumen, there were no distressing symptoms. If, however, the stenosis occluded the trachea to one fourth of its normal circumference stridor and respiratory infection would follow.

4. *Plastic Prosthesis.*—In a series of dogs, a plastic tube prosthesis of Ivalon sponge made over of a frame of a stainless steel spiral was used for tracheal reconstruction This prosthesis was made in the following way:

First, a layer of Ivalon sponge was rolled over glass or plastic tubing. Then a coil of stainless steel was rolled over the Ivalon. Over this, another layer of Ivalon was rolled. Then, this tube of Ivalon was compressed by gauze and autoclaved. In this way, grafts of the tracheal bifurcation could easily be constructed with the desired angle between the 2-main bronchi. Tl sult

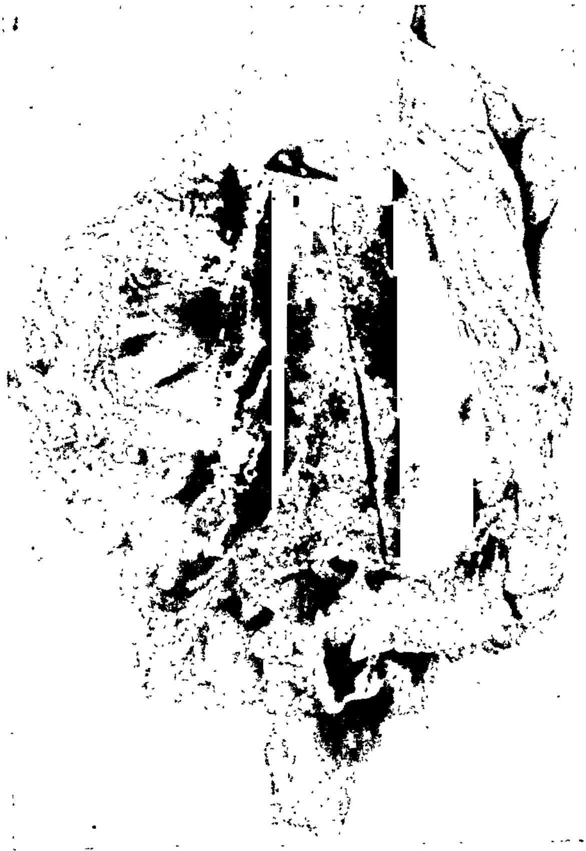


Fig. 5.—Specimen from a bifurcation prosthesis of reinforced Ivalon sponge lying loose in an abscess formation.

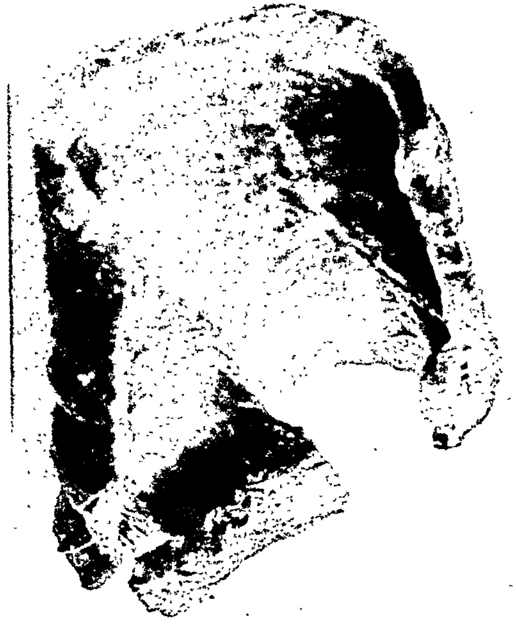


Fig. 6.—Another bifurcation prosthesis which, after 10 months, caused death. It was lying loose in an abscess having perforated the esophagus.

right main bronchus is suggested as the best method to use whenever possible. The left main bronchus of the dog is, however, not suitable for an end-to-side anastomosis.

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with this prosthesis was excellent. However, there was no long-term survival, although the longest survival was 12 months. After a period of 6 to 12 months, this plastic prosthesis either caused a pressure necrosis with perforation of a big artery or oesophagus, or there was an inflammation around the prosthesis so that the prosthesis was actually "floating" in an abscess (Figs. 5, 6, 7). Although some of these dogs seemed in excellent condition up to one year, they then died either of hemoptysis, due to perforation, or infectious complications. Reconstruction of the tracheal bifurcation seems to be especially difficult as it is difficult to obtain the exact angle of the main bronchi; if this angle is not ideal, pressure necrosis will occur at the distal suture line.

DISCUSSION

Reconstruction of the carina with plastic tubes of Ivalon, reinforced with a stainless steel coil, has given a very disappointing result. Although the dogs recovered from the operation and seemed to go through an uneventful recovery for some months, all died within a year. The cause of death was pressure necrosis of arteries or esophagus, or late infections. It was especially difficult to obtain a correct angle between the main bronchi to prevent the distal suture line from pressure necrosis. Therefore, homotransplants were tried but they became necrotic. An autograft of a main bronchus also necrotized when used for tracheal reconstruction. With the very good clinical experience we have from bronchotracheal anastomosis, it is evident that a main bronchus connected with its lung and receiving its blood supply through the pulmonary artery may be used for tracheal reconstruction. In dogs it is easy to resect the lower 12 tracheal rings and do an end-to-end anastomosis of the right main bronchus and trachea. If too great a tension is exerted on the suture line, a stricture formation at the anastomosis will result. In one clinical case of right-sided bronchogenic carcinoma, the left main bronchus was anastomosed to the trachea and after that a right pneumonectomy was performed which included the lower part of trachea and the first part of the left main bronchus. Considerable tension on the suture line resulted as the aorta prevented mobilization of the left main bronchus and a stricture developed. The right main bronchus can be mobilized higher without undue tension on the suture line. All anastomoses were made using 3-0 chromic catgut on atraumatic needles. In an earlier work on the bronchotracheal anastomosis, we found that silk sutures around an anastomosis caused suture granulomas with stricture formation. Catgut will diminish the incidence of suture granulomas considerably and is therefore routinely used for bronchial closure and anastomosis.

SUMMARY

An experimental study of the reconstruction of trachea and its bifurcation is reported. The use of Ivalon prostheses showed good early results but within one year all dogs died due to complications, such as pressure necrosis or infection. A direct anastomosis of the right main bronchus to the trachea, end-to-end, followed by an end-to-side anastomosis of the left main bronchus to the

Obviously, the only hope of reaching an understanding of these lesions is to "catch" such anomalies in the process of formation. With this in view the writer examined part of the large collection of sectioned human embryos in the Carnegie Laboratory of Embryology in Baltimore.* By great good fortune, cysts were found in the upper lobes of a 31 mm. human embryo, and an accessory pulmonary artery in a slightly older 41 mm. fetus.

LUNG CYSTS IN AN EMBRYO OF HORIZON XXIII

The 31 mm. male embryo shown in Fig. 1 is a perfect specimen obtained at operation for tubal pregnancy on a 31-year-old Negro woman. The recorded menstrual age of the embryo was 9 weeks, and the ovulatory age of this horizon was 46 to 48 days (Streeter[20c]). The only malformation observed by the writer in this embryo was a pair of lung cysts.

Fig. 2 is a transverse section through the thorax in the region of the superior mediastinum. The lungs are in the so-called glandular period of development, characterized by early dichotomous budding of the bronchial trees. Branches are lined solely by pseudocolumnar epithelium having several layers of nuclei. At this stage the lungs do not fill the pleural cavities and lie mostly behind the heart.

A single cyst was present on the ventral (anterior) margin of each upper lobe. These were symmetrically placed on right and left lobes and represent an enlargement of a terminal bud of the anterior segmental bronchus. The right one (arrow, Fig. 2) runs through 34 sections 10μ thick (thought to be nearer 12μ) and therefore measures approximately 0.4 mm. in diameter. The patent stalk to which it is appended runs through only 6 sections.

The cyst of the left upper lobe is shown in Fig. 3, enlarged 100 times. It runs through 42 sections and measures approximately 0.5 mm. in diameter. The stalk to which it is attached runs through only 4 sections. If the stalk of the left cyst is patent, it is just barely so. (The section photographed passes to one side of the lumen.) The photographs reveal the fact that both cysts are under fluid pressure; for, proximally, the epithelium of each is cuboidal, and distally, it is flattened.

From a mechanical standpoint the explanation might be sought in the narrowing of the necks of the terminal bronchial buds, in accordance with the Laplace law of pressures in cylindrical tubes (Burton[4]). But, strangely enough, this vase-shaped form of the growing bronchial buds is the prevailing pattern (Fig. 4). Also, these cysts have developed before any smooth muscle fibers have formed around the bronchi. Nor can one attribute the formation of these cysts to any ischemic condition, for the bronchial epithelium is surrounded by an abundant plexus of capillaries (see Fig. 3). Aside from these categories it is difficult to find an explanation. Naturally one asks why cysts do not appear in other portions of these lungs. This raises the question of time specificity in teratogenesis (Wilson[24]). Since there are critical periods in

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*For this privilege the writer is much indebted to Dr James D. Ebert, Director of the Laboratory.

BRONCHOGENIC CYSTS AND THE THEORY OF INTRALOBAR SEQUESTRATION: NEW EMBRYOLOGIC DATA

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SOLITARY or multilocular cysts of the lungs, filled with air or watery fluid and lined with bronchial epithelium, have long been recognized as congenital malformations because, on rare occasions, they have been found in premature and newborn infants. But, so far as the writer is aware, they have never been identified in embryos or in early fetal stages. The problem is further complicated by the frequent presence of infection and in the lower lobes, by the coincidental occurrence of a systemic pulmonary artery. For these reasons the debate still continues as to whether such cysts represent early developmental anomalies or must be attributed to environmental changes of the later fetal and postnatal periods.

For those who hold the former view the most popular theory (Ravitch[17]) seems to be that developed by Pryce and his associates,[15, 16] namely, that cysts represent "sequestered" masses within the lung, comparable to "sequestered" masses of pulmonary tissue found outside the lung. Both are conceived as arising from "traction on the capillary nets of the developing (bronchial) tree by aberrant arteries that anchor the mass while the lung is undergoing developmental shifts." Certainly, the relative frequency with which cysts and accessory pulmonary arteries occur together in patients requiring pulmonary excisions has to be explained; but it does not follow that the two must stand in the relation of cause and effect—especially in view of the fact that they may occur separately. Nor can cysts of the upper lobes be explained by this hypothesis. Nevertheless, all who have wrestled with this problem are indebted to Dr. Pryce and his associates for having recognized the relatively frequent association of these two anomalies in patients and for having presented a theory that has stimulated so much discussion.

The two principal objections to the theory are (1) that it is not consistent with what is known about developmental processes in human and other mammalian embryos (Boyden[1]), and (2) that intra- and extralobar sequestration (lower accessory lung) differ from each other in at least seven ways and therefore "are not related by any mechanism which involves traction by the systemic aberrant artery" (R. Abbey Smith[18]).

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development when certain agents can interfere with development, it may be conjectured that at a time when these particular buds of the anterior segment were growing fastest (*B^2b*, Fig. 4) something interfered with the metabolism of the embryo Once an accumulation of fluid started, the narrow neck could act as a ball valve, ballooning up the terminal segment. In a tubal pregnancy, such as this, the ectopic position of the chorionic vesicle might be a determining factor; but similar interference could arise in normally implanted embryos, witness the experimental production of agenesis of the lung in fetuses of rats whose mothers have been deprived of vitamin A. (See discussion in article by Thomas and Boyden[21]).

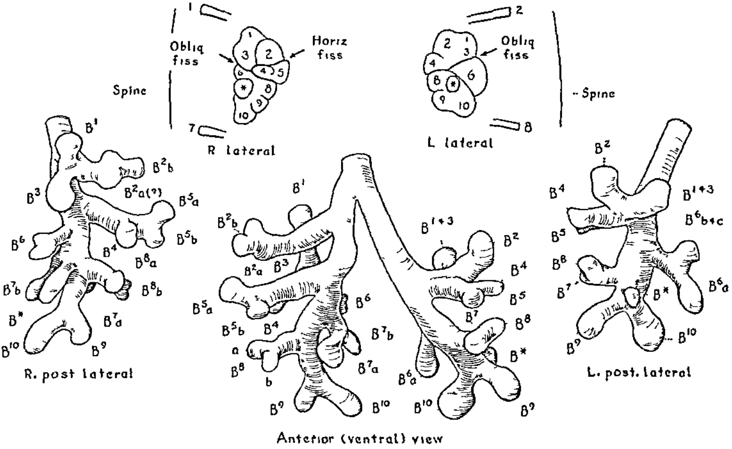

Fig 4—Wax model of the bronchial tree of a 13 mm human embryo of Horizon XVII (After Fig 108 of Boyden's Segmental Anatomy of the Lungs, Blakiston Div., McGraw-Hill Book Co, 1955) *B^1* to *B^{10}*, developing segmental bronchi.

Be that as it may, these cysts could well have developed into solitary bullae which years later could have ruptured spontaneously. If more buds had been involved, these in turn could have compressed adjacent segments and thus have prevented the formation of alveoli. Eventually the whole lobe might have become a mass of cysts (See cases of balloon cysts of infants

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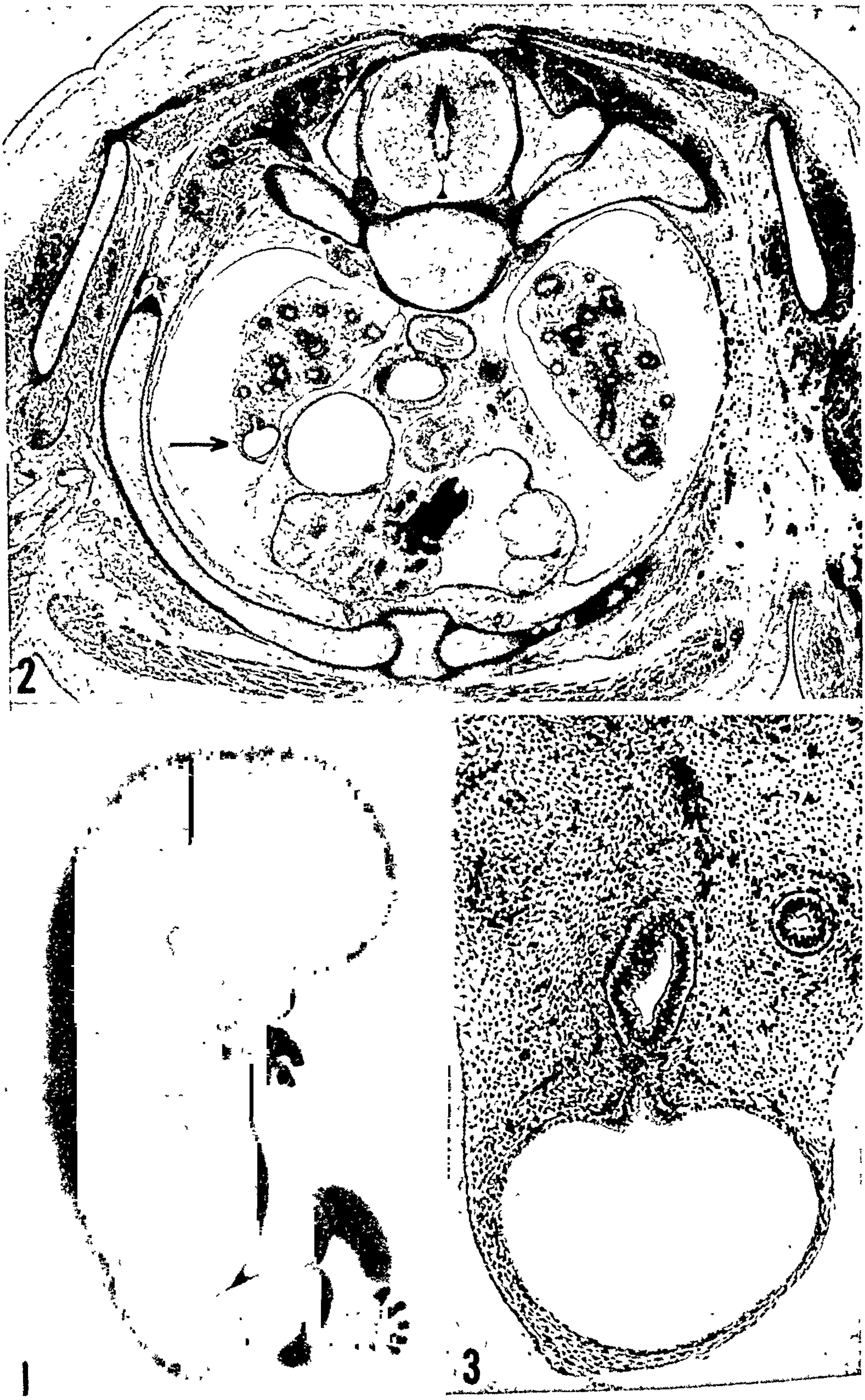
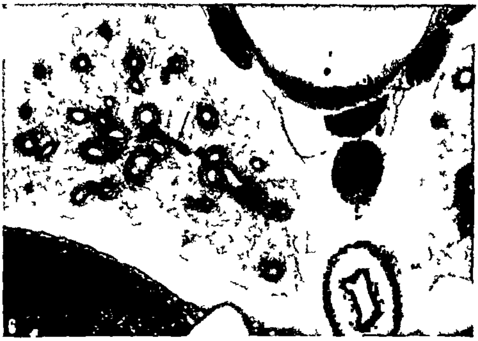
Fig. 1—Photograph of a 31 mm human embryo (Carnegie Coll, No 9226) which contained a symmetrical pair of lung cysts (×3)

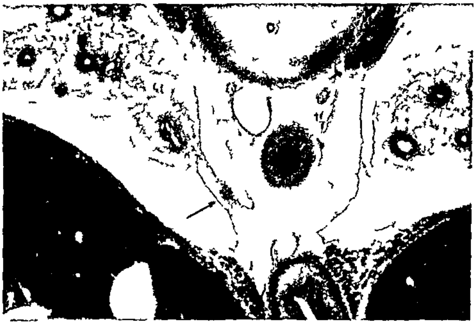
Fig 2—Transverse section through the superior mediastinum of embryo No 9226 (×15) The arrow points to a cyst on the anterior margin of the right upper lobe This is a distended terminal bronchial bud of the anterior segment. Since the left lung is normally smaller than the right, the corresponding cyst on the left side appeared at a slightly lower level (see Fig 3).

Fig. 3—The corresponding cyst on the anterior margin of the left upper lobe (×100) The section passes slightly to one side of the lumen connecting the distended terminal bud with its parent bronchus



Figs. 1-3.—*For legends see opposite page.*





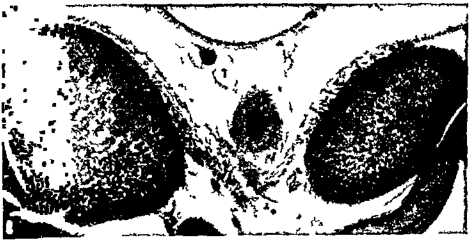


Fig 6 —Transverse section through the right lung of Embryo No 6361 at the level where the accessory artery bends downward (cf top level of Fig 5) The systemic pulmonary artery is indicated by an arrow (×22 reduced ¼)

Fig 7 —Transverse section of Embryo No 6361 showing accessory artery (see arrow) ascending to the lung through the pulmonary ligament (×22 reduced ¼)

Fig 8 —Transverse section of Embryo No 6361 showing accessory artery (see arrow) coursing between the leaves of the crus of the diaphragm

occupying the entire middle lobe, or the entire right lower lobe, or just the superior segment with compression of the rest of the right lower lobe (Potts and Riker,[11] Burnett and Caswell,[5] and others too numerous to mention).

Incidentally, it should be noted that no accessory pulmonary artery was present in this embryo, nor in the infant cases just mentioned. This brings us to description of the first case of a systemic pulmonary artery observed in early fetal stages.

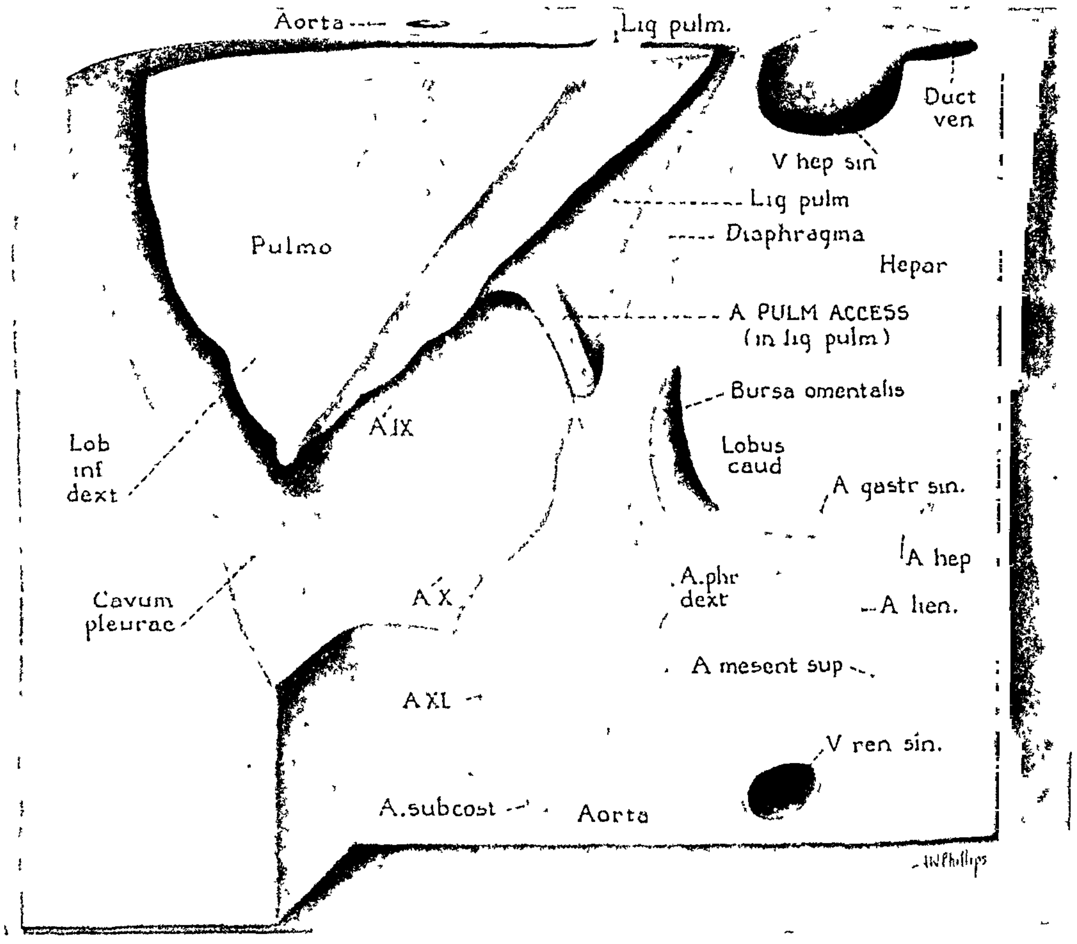


Fig. 5.—Graphic sagittal reconstruction of a 41 mm. human embryo (Carnegie Coll., No 6361 [×30]) showing the topographic relations of an accessory pulmonary artery of aortic origin. The principal plane of section is just to the right of the aorta. On the viewer's left, the plane of section lies parallel to the first plane but cuts through the middle of the right lung.

THE ACCESSORY PULMONARY ARTERY

A systemic pulmonary artery arising from the aorta at the level of the tenth intercostal artery, and supplying the peripheral bronchi of the right posterior basal segment, was found in a young fetus of the Carnegie Collection having a C.R. length of 41 mm. It is of interest to anatomists that this was obtained by Dr. Lawrence Wharton at the hysterectomy of a 32-year-old white woman, and that the embryo was dissected out personally by Dr. Streeter to whom we are indebted for so many *Contributions to Embryology*.

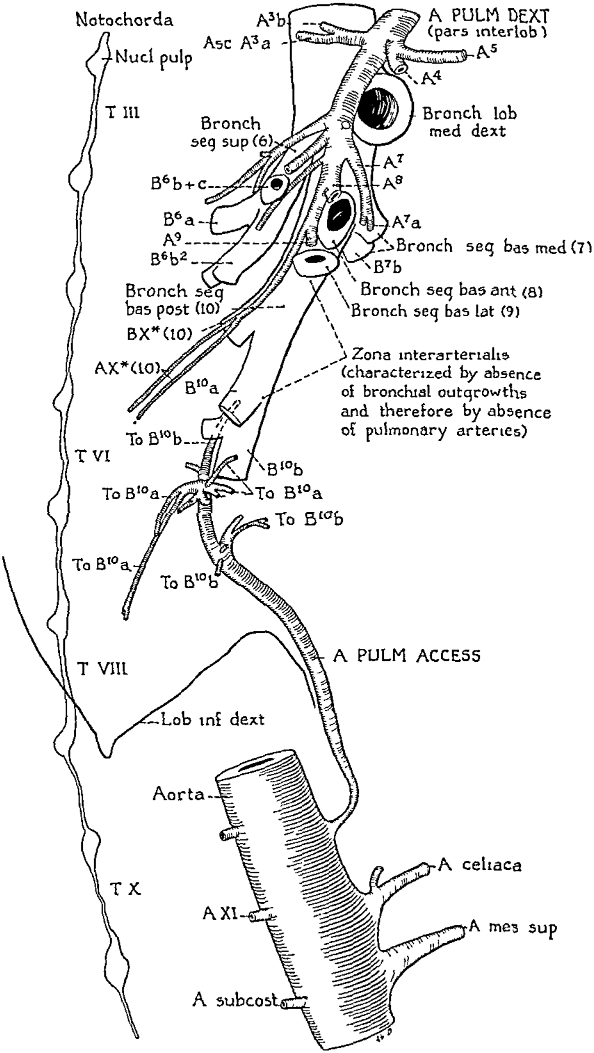


Fig 9 —For legend see opposite page

The patient had had one miscarriage 8 years previously, and 2 normal births at term. Implantation was on the anterior wall of the uterus, and there was "a slight amount of blood in the decidual cavity."

The portion of the embryo containing the artery and adnexa was reconstructed graphically by the author and rendered in three dimensions by Miss Jessie Phillips. The 3 mm. block shown in Fig. 5 represents 60 transverse sections of 50μ each. After the artery leaves the ventral side of the aorta it courses ventralward through the leaves of the diaphragm (see Fig. 8, arrow). (Presumably, therefore, it would have emerged from the diaphragm if the embryo had reached maturity.) Then it enters the free margin of the right pulmonary ligament, from which it bulges (see level of guide line labeled, *A. PULM. ACCESS.*, Fig. 5). Just above this level it is concealed within the broader portion of the pulmonary ligament (Fig. 7). Shortly afterward it leaves the ligament to invade the right lower lobe of the lung. At the height indicated in Figs. 5 and 6 it sprays out and showers down like a Roman candle to supply the lower branches of the posterior basal segment—specifically $B^{10}a$ and $B^{10}b$ (Boyden[2]). At the same level (Fig. 9), it sends smaller branches upward to supply upper rami of B^{10}.

Fig. 9 represents a graphic reconstruction of all pulmonary arteries of the right lung below the level of the upper lobe bronchus. Only the arteries of the accessory subsuperior bronchus $BX^{*}\ (10)$,[19] and those of the aortic pulmonary artery have been drawn in their entirety—that is, so far as they could be traced.

Five principal observations may be made. First, the peripheral portions of both pulmonary arteries are in process of formation. They can be traced only so far as smooth muscle has formed around them. Beyond this point—which is well behind the terminal bronchial buds—they are lost in a minute capillary or (arteriolar) plexus. That is why the specific segmental arteries of an embryo cannot be identified as such, much before the stage of Horizon XIX (Wells and Boyden,[23] Plate 4 and text).

Second, the branches of the aortic pulmonary artery resemble closely those of the right pulmonary artery—both in their structure and in their spatial relations to the bronchi they supply—differing importantly only in origin and in course, but not in structure.

Third, the unbranched portion of the posterior basal bronchus is unaccompanied by any artery. In other words, the posterior basal artery is absent as such except for the small artery, $AX^{*}\ (10)$, which supplies the accessory subsuperior bronchus. The aortic pulmonary, which is taking its place, does not extend beyond the uppermost budding branches of the posterior basal bronchus. There is thus a "no man's land" or interarterial zone which

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Fig. 9.—Graphic reconstruction of the whole pulmonary arterial system of the right lung below the level of the upper lobe bronchus (×30; reduced ½). The upper half of the figure shows the definitive right pulmonary artery. Only the arterial rami accompanying the accessory subsuperior bronchus, $AX^{*}\ (10)$, have been carried as far as they can be traced. The other segmental arteries are shown as cut off at a higher level. Normally, $AX^{*}\ (10)$ is a branch of the posterior basal segmental artery (A^{10}) but, in this embryo, A^{10} is a branch of the aortic pulmonary artery. The lower half of the figure shows the course and rami of this accessory pulmonary artery. *T III – T X*, levels of thoracic vertebrae, between the nuclei pulposi.

DISCUSSION

The sequence of events described above affords a general explanation of why Pryce's theory of the "capture" of bronchial buds by "anchored" arteries seems not to fit the facts of development. If traction on the bulbous tips of the posterior basal bronchus had occurred it should have been revealed at an earlier period; for at this stage of development, in this embryo, it is too late. Yet it has not occurred and the reason is obvious. The nature of embryonic shifts is such that no traction is exerted, even though an artery in the course of development meets a given bronchus at an angle (see Fig. 5). Shifts represent differential rates of growth with corresponding adjustments in the position of vascular trunks. If it were not so, no embryo would ever reach maturity.

Let us now examine the details of Pryce's theory, so far as they relate to embryos. Essential to his hypothesis is the a priori reasoning that "the earlier these abnormal vessels begin the greater is the extent of their distribution in the lung." Ergo, . . . "by referring to the embryonic bronchial tree at different stages it is possible to date their origin. Thus, the artery in Benecke's case II (1905) supplies the two sequestrated lungs and arises at 3 mm. . . . In cases II and VII of Pryce it supplies the posterior basal sector and arises about the 10-14 mm. stage" (Pryce,[15] p. 463; see also Fig. 24, Pryce and others[16]). This deduction seems quite plausible until one examines the topography of a 13 mm embryo (the lungs of which are shown in Fig. 4). Then it appears that an aberrant artery, stemming from the aorta just above the diaphragm, must have had to arise at about the level of the seventh thoracic vertebra in order to maintain its relation to the downward migrating celiac artery (see table of positions of celiac artery in human embryos, Evans,[8] p. 648). Second, it would have had to grow up through differentiating tissues to about the level of the fourth thoracic vertebra, in order to invade the hilum of the lung and descend through the lower lobe to reach the posterior basal bronchi. This is unrealistic. But, accepting the hypothesis for the moment, why then should it not have captured the whole lower lobe?

Another untenable embryonic premise is presented in the article of 1947 (Pryce,[16] p. 23). There the authors state that "the reason it is considered that traction by such delicate structures as embryonic blood-vessels, which are essentially capillary, can lead to amputation of bulbous tips of the developing bronchial tree is that the latter are also fragile, being composed only of thin epithelium embedded in mesenchyme." Unfortunately this is far from being a correct description. Even in very early stages the "bulbous tips" are massive bulbs consisting of a thick pseudocolumnar epithelium containing several rows of nuclei (cf. Streeter,[20b] Fig. 4, p. 146), and they never become less massive

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right lung of a 7-year-old white girl. These vessels of aortic origin entered the mediastinal surface of the lung at different levels. The lung had the structure of a left lung and it was drained by an anomalous pulmonary vein which entered the inferior vena cava. When injected with the superb technique developed by Dr Liebow, the systemic arteries were found to supply portions of the lingular segment, the medial-, anterior-, and lateral-basal segments, and all of the posterior basal segment. The rest of the lung was supplied by the definitive pulmonary artery All of the accessory arteries, except the lingular, swept laterally from the posteroinferior angle of the lower lobe As they approached the bronchi "the branches of the artery became *recurved*, proceeding laterally in close relationship with the bronchi, as is typical of pulmonary arteries. None of these had the spiral anastomosing characteristics of a bronchial artery, despite their aortic origin There was no overlap in the territory supplied by the two sets of arteries" No cysts were present in this lung.

is devoid of arteries. To be sure, the blastema of this segmental bronchus contains minute capillaries which, presumably, will be connected with bronchial arteries; but these are not to be confused with the differentiating strands of the pulmonary arteries which take up a position that is posterolateral to the respective bronchi and lie well outside the blastema.

Fourth, there is no evidence of any extraneous factor, such as compression or atrophy, to account for the absence of the definitive posterior basal branch (A^{10}) of the right pulmonary artery. The explanation would seem to be a much simpler one—namely, that since the growing tips of pulmonary arteries terminate in the peripheral vascular plexus of the lung, the original area of distribution of the right pulmonary and the accessory artery has been moved apart by interstitial growth of the trunk of the posterior basal bronchus. (For elaboration of this idea, see "Discussion.")

Fifth, the most striking feature of the accessory artery is its relatively great length and its nearly vertical course. This, however, is only a temporary situation. Had the fetus lived, and had the lung grown down to its definitive position, the artery would have become nearly horizontal and would have resembled both in position and distribution that of Kinsella's case (see Boyden,[2] Figs. 120-122).

One may now ask, what was the original position of this systemic pulmonary artery. In the 4 to 5 mm. stage when it first appeared (age 28 to 30 days, Horizons XIII-XIV) it must have arisen at the level of one of the upper cervical vertebrae and have had the appearance of a more or less horizontal branch passing from the ventral side of the left aorta to the closely adjacent visceral plexus surrounding both the esophagus and trachea and the two lung buds.* Tobin has drawn such a hypothetical branch in his discussion of the origin of bronchial arteries (Tobin,[22] Fig. 1). The stage in question is that in which the lung buds and esophagus are closest together. At any later time it would be impossible for a ventral branch of the aorta to reach the bottom of the lung without traveling a most circuitous course. At this early stage, the celiac artery lies at about the level of the seventh cervical vertebra (Evans,[8] p. 648). Between this stage and the stage of the 41 mm. embryo (see Fig. 5), the accessory pulmonary artery and the visceral arteries below it will shift their points of origin along the ventral side of aorta faster than the lung grows downward (for discussion of this mechanism see Evans,[8] p. 648).

In summary, the following sequence of events can be established: first, the accessory pulmonary artery is approximately horizontal, lying in the region of the upper cervical vertebrae; second, its aortic end shifts with the visceral arteries, down along the ventral side of the aorta, until the artery becomes nearly vertical; third, the lung catches up with it and the artery to the base becomes nearly horizontal again. This shift in position explains why the upper portion of the accessory artery, in the stage shown in Fig. 5, meets the posterior segmental bronchus at an angle and then curves downward to supply its branches.†

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*Cf. Congdon[7]; Buell[3]; Streeter[20a]; and Neill[12].

†A similar relationship of systemic arteries to bronchi has been reported recently by Halasz, Halloran, and Liebow.[9] They encountered six accessory pulmonary arteries in the

(Cont'd on opposite page.)

others) or in cases where there are five or six aortic pulmonary arteries supplying peripheral branches of the lingular and basal segments (Halasz and associates[9]) Indeed, it is quite possible—witness the 57 cases collected by Cole, Alley and Jones[6]—that systemic arteries to the lower lobes are more numerous than has been suspected, and that many are never seen, surgically, because they are not associated with diseases of the lung or other circumstances requiring surgical intervention

Because of these considerations, the writer suggests that until there is evidence to the contrary one must fall back upon the theory of coincidental occurrence of lung cysts and systemic pulmonary arteries An analogy to this may be found in cases of agenesis of the lung This lesion can be produced experimentally in fetal rats by feeding the mother with a diet deficient in vitamin A Frequently associated with this condition are anomalies of the aortic arches (Wilson and Warkany[25]) Yet either condition can be present without the other

SUMMARY

Lung cysts developing spontaneously in a 31 mm human embryo, and a systemic pulmonary artery supplying the posterior basal segment of a 41 mm fetus have been discovered in sectioned human embryos of the Carnegie Collection These anomalies have been described and discussed in relation to Pryce's theory of intralobar sequestration It is concluded that a hypothesis of coincidental occurrence of these anomalies in certain patients is still all that can be warranted by the facts at our disposal

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(Figs. 3 and 6) until the glandular period of growth is completed. How, then, is it mechanically possible for such bulbs to be "captured" by vessels that "are essentially capillary"?

A third source of confusion is the belief that there is competition for the lung buds on the part of the two arteries (Pryce and others,[16] p. 22); or that the persistence of the systemic artery is due to some breakdown in the formation of the pulmonary artery (Smith[18]). What really happens is that very early in development the trachea, the paired bronchial buds, and the esophagus are surrounded by a visceral plexus of capillaries. Soon this system is tapped by incipient pulmonary arteries and veins and by any aberrant systemic channel (arterial or venous) which happens to be present. It is the persistence of these latter connections which produces the varied vascular anomalies reported in the literature. When such an accessory arterial channel persists, it merely takes over the bronchial buds in its vicinity while the incipient pulmonary artery takes over the rest. These zones may then be moved apart by interstitial growth of the larger bronchi so that a "no-man's land" is left between them.* Neither artery has failed or overcome the other; each merely restricts itself to the bronchi with which it is in contact. Meanwhile, the proximal portions of both the typical and the accessory pulmonary arteries begin to develop smooth muscle coats—dense tunicae mediae which distinguish them as arteries. But this zone of differentiation always remains a certain distance behind the growing bronchial buds. Presumably also, in time, the wall of the systemic artery may develop a wall like that of the pulmonary artery which is predominantly composed of elastic tissue.

Finally, there are the numerous reasons why intra- and extrapulmonary sequestration should be considered as having a different etiology. These have been covered so thoroughly and critically by R. Abbey Smith[18] that this ground need not be harrowed again.

How then may the frequent concurrence of lung cysts and systemic pulmonary arteries be explained? A very attractive theory is the one presented by R. Abbey Smith[18]—namely, that after birth that portion of the lung which is subjected to systemic blood pressure may undergo cystic and fibrous degeneration. The strongest evidence that can be summoned in support of this theory is Potter's observation[13] that intralobar sequestration has not yet been detected in 10,000 post-mortem examinations of children under one year of age. But, as Dr. Potter would be the first to point out, failure to demonstrate a condition in innumerable autopsies does not establish the fact that it cannot occur. It merely means that if it does occur it is extremely uncommon. Also, it is probably that conditions which are not immediately lethal seem to be recorded less often in autopsies than one would anticipate from their occurrence in the general population. Finally, Smith's theory would require explanation of the absence of associated cysts in mature individuals in which there is a stout systemic pulmonary artery (e.g., cases by McCotter,[11] Pryce,[15] Kinsella,[10] among

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*Later, perhaps, this interarterial zone may be bridged by bronchial arteries and their anastomotic connections with the pulmonary arteries.

GRANULOMATA OF THE MEDIASTINUM SURGICALLY TREATED AND FOLLOWED UP TO NINE YEARS

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GRANULOMATA of the mediastinum assume surgical significance from two aspects First, they may present as mediastinal tumors of undetermined etiology for which exploratory thoracotomy is indicated for diagnostic purposes [1, 2] Second, they occasionally encroach upon vital neighboring structures and cause complications involving the trachea, bronchus, lung, esophagus, or superior vena cava [3, 10]

In the early reports, these lesions were designated "tuberculomata †[2, 11] Actually, they are conglomerate caseous granulomata of lymph nodes pathologically, often described "consistent with tuberculosis " Although grossly and histologically they are typical of tuberculosis in most of the cases, the etiology cannot be determined with accuracy because organisms are often not demonstrable in the lesions Fungi are also know to produce similar lesions A more accurate designation is therefore "mediastinal granuloma "

There have been several reports of these granulomatous mediastinal tumors being removed either entirely or partially These cases were summarized by Kunkel, Clagett, and McDonald,[12] and they reported 32 cases proved at thoracotomy including 16 of their own Subsequently, Perdue and Guilfoil[13] reported 3 additional cases Although these granulomata are considered to be of tuberculous or fungus origin, there is no long term follow up of these patients to indicate that they remain free of further manifestations of active disease for significant lengths of time This report deals with 19 patients who were subjected to exploratory thoracotomy for mediastinal tumors which proved to be granulomata They have been followed for periods up to 9 years

Clinical Aspects—The majority of these patients were discovered to have mediastinal masses on routine roentgenological examination of the chest Two patients reported having had some discomfort in the chest One patient had pain in right shoulder and right arm (Case No 14) Two patients had a slight

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†Tuberculomata are firm discrete round lesions On section they may present a laminated appearance with concentric rings alternating with areas of calcification Microscopically one invariably finds areas of caseation Should they present an area of central cavitation they should not be designated as true tuberculomata

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10	B G M 26	T 0 H – C –	5 × 3 cm rt paratracheal	Excision	Fibrocaseous granuloma, calcium	SM INH PAS, ambulatory, 1 yr	Asymptomatic	3
11	M S M 48	T + H + C 0	5 × 3 × 8 cm rt. paratracheal	Excision	Granuloma	INH SM, ambulatory, 1 yr	Asymptomatic	1
12	A C V M 21	T 0 H + C 0	3 × 4 cm rt paratracheal	Partial excision	Caseous granuloma, lymph nodes	SM PAS 1 yr, ambulatory	Asymptomatic	1½
13	J J P* M 26	T + H – C –	4 × 6 cm left heart border	Mass excised from left pericardium	Cystic granuloma	SM and PAS 42 days, bed rest 6 mo	Asymptomatic	7
14	C M* M 35	T + H – C –	Rt paratracheal	Partially excised	Fibrocaseous granuloma	SM and PAS 4 mo	Asymptomatic	6½
15	W J S M 19	T + H 0 C 0	Rt paratracheal	Excised	Fibrocaseous granuloma	None	Bilat apical TBC 3 mo postop, then treated with SM PAS and bed rest for 8 mo, now asymptomatic	1½
16	F B S** M 35	T + H 0 C 0	Midmediastinal, subcarinal	Unsuccessful removal on rt, removed from left at 2nd operation	Fibrocaseous granuloma	None	Developed cavitary TBC *RUL* 4 yr postop, under treatment	4
17	J C R M 29	T + H – C –	2.5 cm rt paratracheal	Excision	Fibrocaseous lymphadenitis	None	Asymptomatic	4
18	M L M F 22	T 0 H 0 C 0	3.5 × 2.5 cm rt tracheal	Biopsy	Granuloma, lymph node	None	Asymptomatic	2
19	H M M 27	T 0 H + C 0	4 × 3 cm rt paratracheal	Incomplete excision	Granuloma, histoplasmosis	None	Asymptomatic	2½

Key to abbreviations T = Tuberculin H = Histoplasmin C = Coccidioidin + = Positive 0 = Negative – = Not reported
*Reported by Storey and Lyons[3]
**Reported by Lyons and Storey[4]

Table I. Patients With Mediastinal Granulomata Subjected to Surgery

CASE NO.	SEX AGE	SKIN TESTS	LOCATION AND SIZE	SURGERY	PATHOLOGY	POSTOP. THERAPY	FOLLOW-UP	TIME FOLLOWED (YEARS)
1	E.W. L. M 20	T - 0 H - - C - -	4 cm. rt. ant. sup. mediastinal	Excised	Cystic caseous granuloma of node	Bed rest 5 mo.; no drugs	Asymptomatic	7
2	F. W. B. M 19	T - 0 H - 0 C - 0	7 × 5 cm. rt. post. sup. mediastinal	Excised	Caseous calcified granuloma of node	SM 6 mo.	Asymptomatic	5
3	W. E. H.* M 25	T - + H - - C - -	3 cm. rt. paratracheal	Excised	Tuberculosis of node; *positive* for AFB (culture)	6 mo. bed rest; SM and PAS 42 days	Asymptomatic	6
4	D. P. G.* M 21	T - 0 H - - C - -	3 cm. rt. paratracheal	Excised	Caseous granuloma of node	Bed rest; SM and PAS 3 mo.	Asymptomatic	6
5	M. A. O'B.* M 24	T - + H - - C - -	5 × 3 × 2 cm. rt. paratracheal	Excised	Caseous granuloma; *positive* for AFB (smear)	Bed rest 3 mo.; no drugs	Asymptomatic	9
6	E. L. D. M 23	T - + H - + C - 0	3 × 4 cm. rt. paratracheal	Excised	Fibrocaseous granuloma	None	Asymptomatic	5
7	S. L. R. M 19	T - + H - 0 C - 0	5 × 5 cm. rt. paratracheal	Lesion eroded into lung; incomplete excision of mass and wedge resection of lung	Fibrocaseous granuloma	SM and PAS; bed rest 6 mo.	Asymptomatic	4
8	D. M. K. M 24	T - + H - 0 C - 0	2.5 cm. defect in esophagus at level of carina	Excised from carina and esophagus	Tuberculosis of node; *positive* for AFB (culture)	SM and PAS; bed rest 8 mo.	Asymptomatic	4
9	A. L. F 21	T - + H - 0 C - 0	3 × 4 cm. left hilar	Biopsy	Tuberculosis of node; *positive* for AFB (culture)	SM and PAS; ambulatory, 1 yr.	Asymptomatic; mass regressed in size	2½

enlarged nodes were felt at or below the hilum of the lung. The lung was frequently adherent to the mass, but it was involved in the mass in only one instance.

The granuloma presented at the left heart border, the subcarinal region, encroaching upon the esophagus from the right just below the bifurcation of the trachea, and at the left hilar region, in one instance each.

Pathologic Aspects.—Grossly, these lesions were lobulated and either solid or cystic. They were hard in some areas and soft in others. Many of the cysts ruptured easily (Fig. 3, *B*). Gross calcification was present in 3. The central portions of these granulomata were yellow or white and contained caseous material of a chalky or cottage-cheese consistency (Fig. 4).

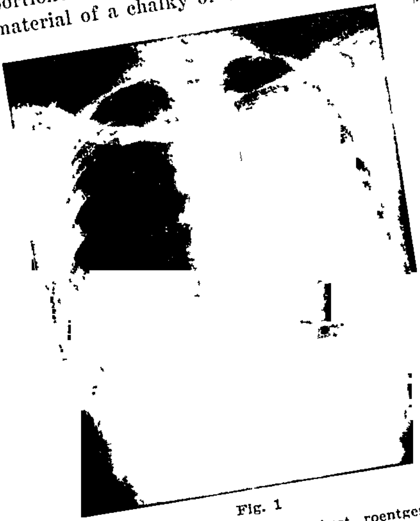

Fig. 1

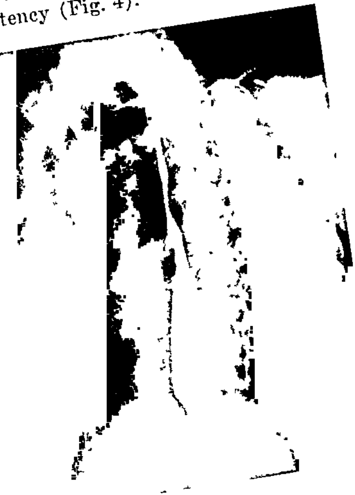

Fig. 2

Fig. 1.—Posteroanterior chest roentgenogram of patient with mediastinal granuloma. Typical roentgen finding of lobulated mass projecting to right of superior mediastinum.

Fig. 2.—Esophagram showing granuloma of mediastinum causing filling defect in esophagus.

All of these lesions were found to be granulomata histologically. Some were described by the pathologist as "fibrocaseous granulomata" and others described simply as "granulomata." Some of the lesions showed small tubercles with epithelioid cells, Langhans' type of giant cells and caseation (Fig. 5). Some revealed no giant cells or epithelioid cells, but were composed mainly of fibrous tissue and inflammatory cells. Nine of these granulomata had remnants of lymph nodes in the walls. The remaining 9 had no lymph node component recognizable grossly or histologically.

All of the lesions were examined microscopically for acid-fast bacilli and 18 were cultured for *Mycobacterium tuberculosis*. Three of the lesions were positive for *Mycobacterium tuberculosis* on culture. One which was negative daily elevation of temperature for 3 weeks prior to admission. Four patients complained of cough, one with hemoptysis, several years previously. One patient (Case No. 15) complained of fever, malaise, and weight loss (subsequently developed overt pulmonary tuberculosis 2 months postoperatively). One patient (Case No. 17) was placed on streptomycin, isonicotinic acid hydrazide (INH), and para-aminosalicylic acid (PAS) therapy after biopsy of a mediastinal tumor showed granuloma; the mass increased in size roentgenologically over the next several months and excision was performed. One patient, who had a partial esophageal obstruction due to granuloma, had dysphagia for 2 years.

Physical examination was noncontributory in each case. Hematological examination, urinalysis, and the other usual laboratory examinations were not helpful in establishing the diagnosis.

Skin tests for tuberculosis, coccidioidomycosis, and histoplasmosis were performed in many of the cases. The results of these tests are tabulated in Table I.

Bronchoscopy was performed in many of the cases. No endotracheal or endobronchial lesion was demonstrated, but extrinsic compression of the trachea was present in one case. Esophagoscopy was negative in the case in which esophagram revealed an extrinsic mass encroaching upon the esophagus.

In none of these cases was there any concomitant lesion visible on the roentgenogram or evident on physical examination which made it possible to conclude that the mediastinal mass was definitely tuberculous or fungous in origin and not a mediastinal tumor of other type.

Roentgenological Aspects.—The majority (15) of these mediastinal granulomata presented as mediastinal masses projecting from the mediastinum to the right above the hilum of the right lung (Fig. 1). Typically, they were situated in the right paratracheal region, were usually lobulated, and occasionally revealed spotty calcification. Tomography was useful in delineating the size, position, and character of these lesions. One of the masses was situated beneath the carina of the trachea, one at the left hilum, and one at the left heart border. One case presented a mediastinal tumor encroaching upon the esophagus, suggesting the possibility of a leiomyoma of the esophagus (Fig. 2). These masses ranged in size from 2.5 cm. to 8 cm. in the largest dimension. In none of these case was there significant hilar adenopathy (one presented as a left hilar mass). In no case was a lesion noted in the lung fields. In one patient (Case No. 14) obstruction of the right subclavian vein was demonstrated by angiography.

Anatomic Aspects.—Fifteen of these granulomata presented in the right paratracheal region beneath the mediastinal pleura, immediately superior to the right hilum. They usually were situated superior to the azygos vein and anterolateral to the trachea, somewhat posterolateral to the superior vena cava. Very often the mass was intimately adherent to the trachea, bronchus, superior vena cava, azygos vein, or pulmonary artery. Occasionally additional

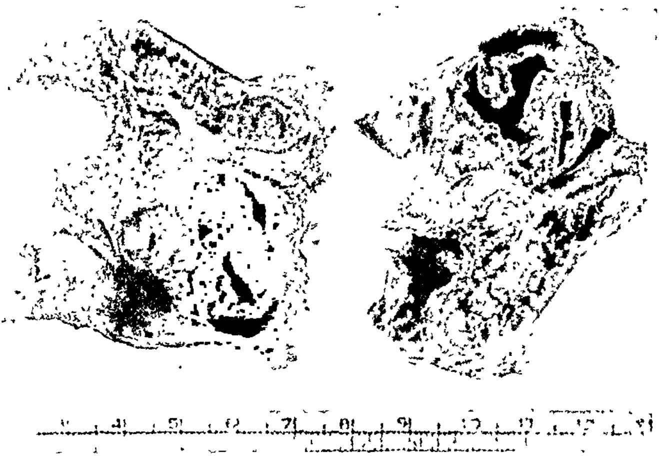

Fig. 4.—*Cut gross specimen of mediastinal granuloma in Fig. 3, B showing cystic nature* and caseous central portions

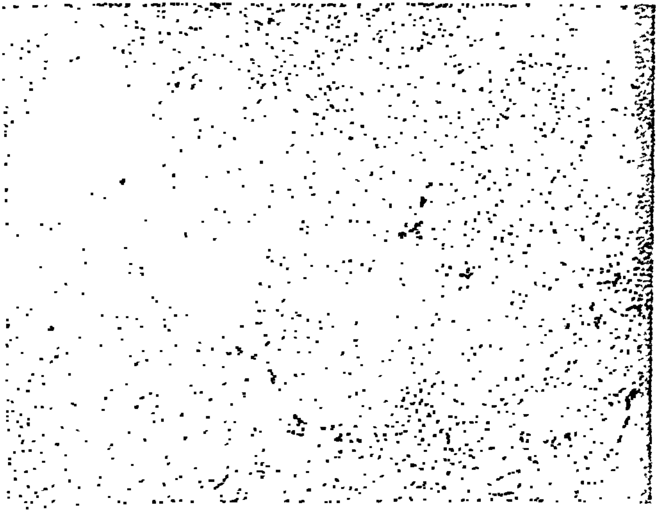

Fig. 5.—Photomicrograph of typical mediastinal granuloma (×50). Giant cells, epithelioid cells, and caseous necrosis are present.

on culture had been positive for acid-fast bacilli microscopically. Many of the granulomata were examined microscopically and cultured for fungi, and one was positive for *Histoplasma capsulatum.*

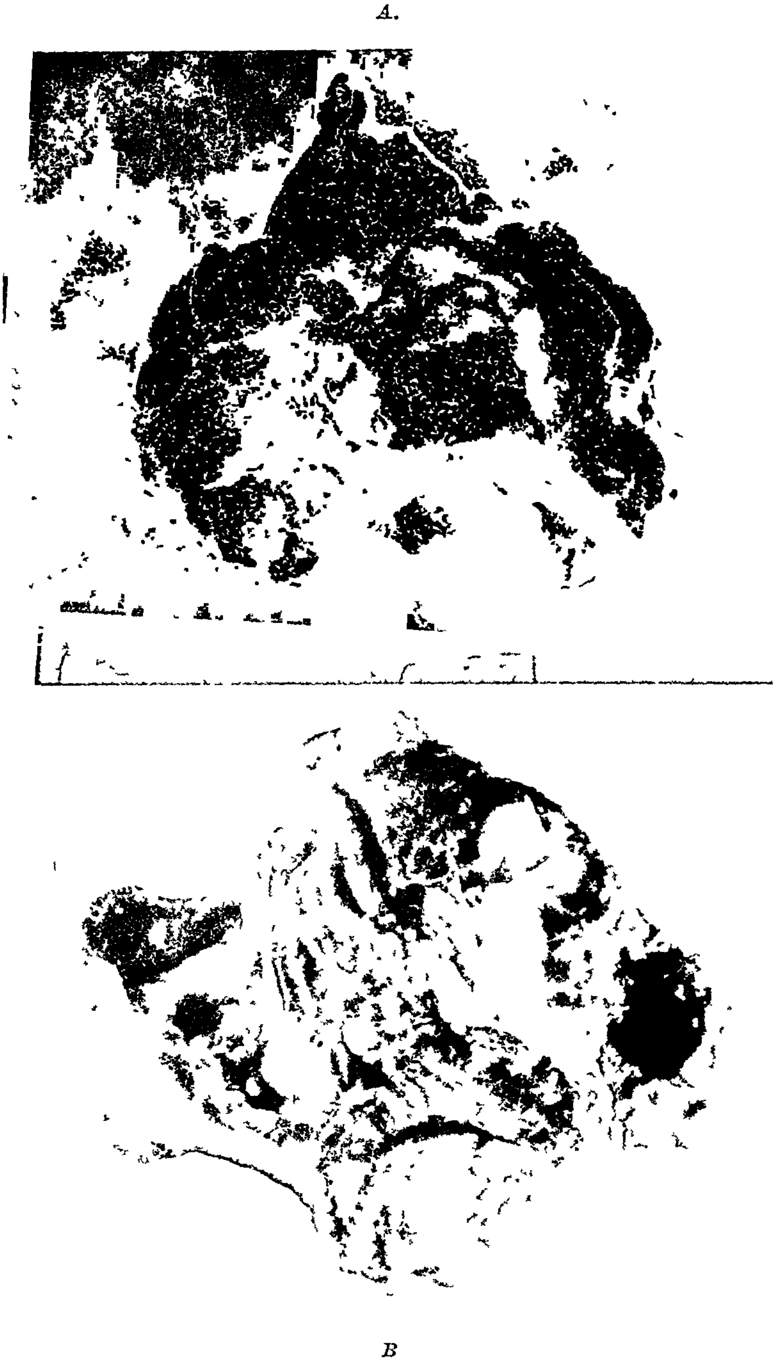

Fig. 3.—*A*, Gross appearance of typical mediastinal granuloma.

B, Gross appearance of a mediastinal granuloma which was partially opened during excision.

Surgical Aspects.—Sixteen of these lesions were approached through a right posterolateral thoracotomy and 2 through a left posterolateral thoracotomy. One was approached at a first stage by right thoracotomy, and because was not given. The postoperative course was uneventful, and the patient was discharged 4 weeks postoperatively. While at home he began to have fever and malaise again 2 months postoperatively and was readmitted to the hospital with bilateral apical pulmonary tuberculosis. Treatment with streptomycin and PAS was then instituted and continued for 8 months. He is asymptomatic at present.

Despite the fact that there was no x-ray or bacteriologic evidence of pulmonary tuberculosis preoperatively, it may be that this patient was suffering from active tuberculosis at the time of thoracotomy. In any event, his resistance to tuberculosis was insufficient to prevent the development of bilateral apical pulmonary lesions. Postoperative antituberculosis chemotherapy may have prevented the development of clinically active pulmonary tuberculosis in this case.

Case No. 16 had a large subcarinal mass removed through a left thoracotomy after previous right thoracotomy had not given adequate exposure for removal of the mass. He received no bed rest or chemotherapy for tuberculosis postoperatively. The lesion pathologically was nonspecific fibrocaseous granuloma [illegible] fast bacilli were demonstrated by smear or culture. The post[illegible] was uneventful and the patient felt entirely well for 4 years [illegible]. He developed cavitary pulmonary tuberculosis in the right [illegible] sputum 4 years postoperatively.

[illegible] has less than average constitutional resistance to tuberculosis [illegible] chemotherapy would probably have suppressed late[illegible] sufficiently to prevent the subsequent development of [illegible] tuberculosis.

DISCUSSION

[illegible] the etiology of these granulomata is similar to that rep[illegible] chronic fibrocaseous granulomata. [illegible] as histoplasmosis by [illegible] demonstration of organisms. These masses are generally assumed to develop [illegible] enlargement of lymph nodes, and 9 of these granulomata exhibited remnants of lymph node histologically. The remainder probably represent completely destroyed [illegible] abscesses which develop following rupture of a caseous lymph node, such as reported by Samson.[illegible]

Exploratory thoracotomy is indicated for all mediastinal tumors of undetermined etiology. [illegible] removal of a mediastinal granuloma in the [illegible]. Removal of the granuloma is indicated to prevent [illegible] of the granuloma [illegible] trachea, bronchus, [illegible] or lung and to prevent [illegible]. If complete excision is frequently [illegible] because of adherence to [illegible] vital structures, removal of as much of [illegible] will reduce the size of [illegible] it was impossible to remove the subcarinal mass at the first operation, a left thoracotomy was performed later to complete the removal. Posterolateral thoracotomy through the fourth interspace gave excellent exposure of the typically placed paratracheal mass. The azygos vein was divided in most cases in which excision of the mass was performed.

Upon exposure of the mass, it was found to be injudicious to attempt removal of the granuloma completely in 6 cases because of marked adherence to the trachea, bronchus, vena cava, or pulmonary artery. Therefore, biopsy only was performed in 2 cases and partial excision of the mass was performed in 4 cases. In the remaining 13 cases, the mass was removed completely. In a number of these cases, the mass was opened during the course of removal and the caseous material was spilled into the thoracic cavity. In one case, the right paratracheal mass was intimately adherent to the right upper lobe of the lung and a wedge of right upper lobe was removed along with a portion of the lesion. Partial excision of the mass in Case No. 14, in which obstruction of the right subclavian vein was shown preoperatively, relieved the venous obstruction.

Postoperative Complications and Treatment.—All of these patients recovered from their thoracotomy without complication. There was no case of empyema or pulmonary infiltration recognizable postoperatively (except for Case No. 15). Eleven patients received some combination of streptomycin, INH and/or PAS for periods ranging from 6 weeks to one year with no postoperative evidence of disease during follow-up periods up to 6 years. Two patients received bed rest only in periods ranging from 3 months to 5 months, and these patients are all well in follow-up periods up to 9 years. Six patients received no postoperative therapy directed toward the prevention of complications or exacerbations of tuberculosis, and 2 of these patients have subsequently developed active pulmonary tuberculosis. One of the patients that received no particular postoperative therapy was demontrated to have *Histoplasma capsulatum* organisms in the specimen, and he has been well for 2½ years following biopsy of the mass.

Follow-up of These Cases.—These cases have been followed up to 9 years, and all have been asymptomatic without evidence of further disease except 2 patients.

Case No. 15 had a history of malaise, fever, night sweats, and a 20-pound weight loss for one month prior to admission. Physical examination revealed no abnormalities except for fever of 103° F. Roentgenogram of the chest was negative except for a right upper mediastinal mass. Skin tests for tuberculosis were positive. During hospitalization, the temperature returned to normal and the patient gained 10 pounds. · Approximately 6 weeks after admission, thoracotomy was performed with a preoperative impression of Hodgkin's disease, and the mediastinal mass was excised. Small nodules were felt deep in the substance of the lung. No biopsy of these was taken because of the depth of the lesions in the lung. The specimen was a fibrocaseous granuloma, and smears and cultures for tuberculosis were negative. Antituberculosis therapy

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these structures. However, because most of these granulomas do not cause serious complications, biopsy alone is sufficient if further excision appears to be contraindicated.

Mediastinal granulomata of tuberculous origin are probably manifestations of postprimary localized mediastinal lymphadenitis. The development of clinical pulmonary tuberculosis in 2 of these 19 patients is probably not a result of the surgical intervention per se, but is a manifestation of a constitutional susceptibility of these individuals to tuberculosis. These individuals may develop exacerbations at any time, since they harbor latent tuberculous foci. Medlar[15] has emphasized that caseous foci contain viable tubercle bacilli that may cause a relapse of disease years later. Because the 2 patients who developed clinical pulmonary tuberculosis did not have antituberculous chemotherapy, we now assume that all of these granulomata which are not demonstrated to be of fungus etiology are of tuberculous etiology although organisms are not demonstrated. Therefore, all of these patients in whom a fungus is not demonstrated in the lesion receive antituberculous chemotherapy routinely for 6 to 12 months. The chemotherapy is given to prevent possible sequelae of contamination of the pleural cavity with the contents of the granulomata during removal and to prevent further exacerbations elsewhere. We prefer to give INH and PAS and reserve streptomycin for possible further use in the future.

CONCLUSIONS

1. Exploratory thoracotomy is necessary for the diagnosis of mediastinal granulomata when there is no other clinical evidence which would indicate the granulomatous nature of these lesions.
2. Removal of a portion of the granuloma for biopsy is essential in every case. Excision of as much of the mass as is feasible without undue risk is accompanied by no further complications than biopsy alone.
3. Postoperative antituberculous therapy for 6 to 12 months should be administered to prevent subsequent spread or reactivation of tuberculosis unless a nontuberculous etiology is definitely established.

The authors wish to express their deepest appreciation to Dr. J. Maxwell Chamberlain, who first advocated exploratory thoracotomy for possible mediastinal granulomata at the U. S. Naval Hospital, St. Albans, who operated upon the first patient, and who gave many valuable suggestions during the preparation of this manuscript. We also wish to thank Dr. Robert Klopstock, Chief of Thoracic Surgery at the U. S. Veterans Hospital, Brooklyn, N. Y., for his suggestions and permission to include his cases (No. 10 and No. 11).

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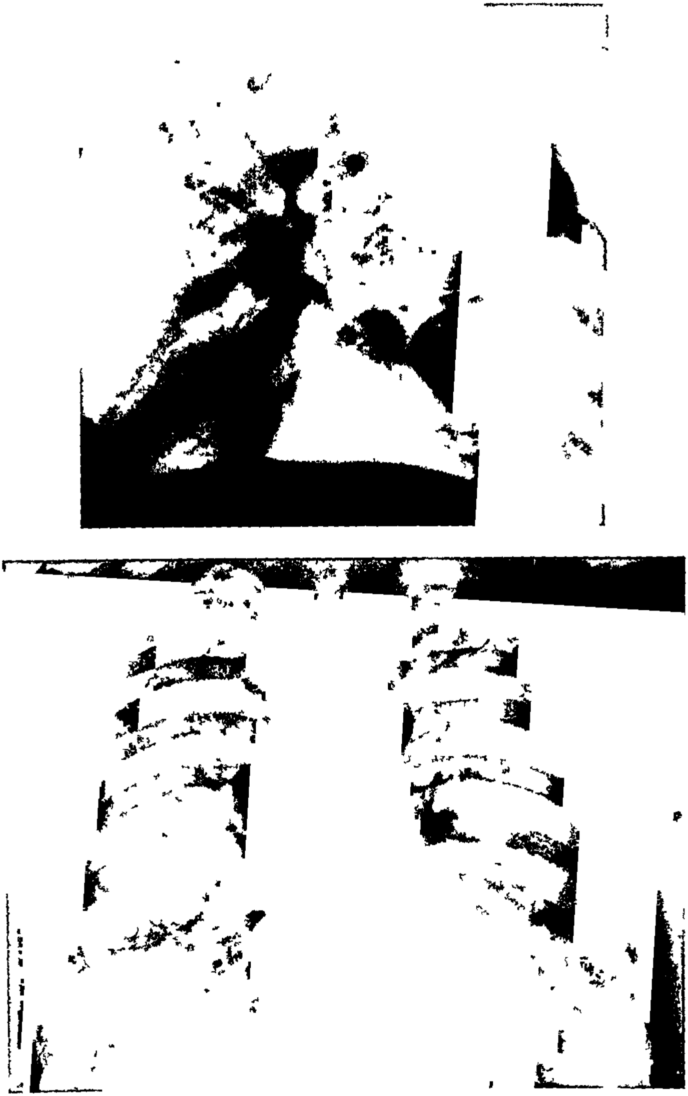

Fig 1.—Frontal and lateral views showing 3 of the 4 tumorlike processes in the left lung

TUMOR-FORMING AMYLOIDOSIS OF THE LUNG

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INCOMPLETE knowledge of the etiology and nature of amyloid degeneration makes classification of amyloid disease difficult. In 1929, Lubarsch[8] suggested a uniform nomenclature and coined the term atypical amyloidosis for those cases deviating from the well-known classical type. Herxheimer and Reinhart[4] suggested a division of amyloidosis into two types, namely generalized and local, while Rosenblum and Kirschbaum[11] classified the condition as primary or idiopathic and as secondary or symptomatic with subdivision of each group into diffuse (typical) and local (atypical) forms. King[5] based his classification on the anatomic distribution and distinguished typical and atypical amyloidosis associated or not associated with other disease. The classification often referred to in the literature is that suggested by Reimann and associates.[10] They recognized three groups in addition to the secondary amyloidosis of chronic infectious diseases, namely, primary atypical amyloidosis, characterized among other things by the absence of any disposing basal disease; nodular, solitary or multiple local amyloidosis (amyloid tumors) and, finally, amyloidosis associated with multiple myeloma. Though nodular amyloidosis is usually primarily atypical, it appears justified to assign amyloid tumors to a special group because of their characteristic pathologic features.

Amyloid tumors have been the subject of research for many years, especially in Germany. Leupold[6] emphasized the value of publishing cases of localized amyloidosis and stressed the importance of descriptions of the tumors in the elucidation of problems bearing on amyloidosis. The factors considered of importance for the development of secondary amyloidosis can nearly always be excluded in nodular amyloidosis. Another essential difference is the fact that amyloid tumors are seldom seen in parenchymatous organs. They show a predilection for striated or smooth muscle, skin, lymph nodes, and, occasionally, for the mucosa of the digestive and urinary tracts. The base of the tongue is also a common site of circumscript nodular amyloidosis. Multiple amyloid tumors in the upper respiratory tract are not uncommon. This form of amyloidosis is rarely seen in the lung parenchyma, and a search of the literature failed to reveal reports of more than 13 cases. All of them have been recently reviewed (Glauser[2]). The pulmonary changes sometimes produced symptoms of pneumonia and pleurisy, sometimes they produced no clinical symptoms and were discovered incidentally at roentgen examination or post

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Fig. 2.—Roentgenogram [illegible] (Jan. [illegible]

mortem. In one case of solitary amyloid tumor, the patient, a man aged 66 (Haynes and co-workers[3]), was operated on because of a presumptive diagnosis of pulmonary tumor. In the case reported below the preoperative diagnosis was secondary lung tumor from a primary tumor elsewhere.

CASE REPORT

K. A., a farmer aged 67 years, was treated conservatively for duodenal ulcer in 1932 and 1933. In 1955, the patient sustained a blow against the left flank with costal infraction (site no longer demonstrable in roentgenogram), otherwise he had always felt well.

He was admitted to the department of thoracic surgery on Aug. 29, 1956, after repeated mild infections of the respiratory tract during the previous 3 to 4 months and a markedly progressive nonproductive cough for 5 weeks. An earlier roentgenogram of the chest (August 17) had shown the presence of at least 3 tumorlike changes in the left lung (Fig. 1). Of these, 2 were located in the lower lobe and one in the upper lobe.

Apart from the troublesome cough, the patient felt well. Routine examination revealed no pathologic palpatory changes of the internal organs or lymph nodes and roentgen examination of the digestive tract, urinary tract, and skeleton showed nothing abnormal. The blood values and the blood picture were also normal. The sedimentation rate was 16 mm. per hour.

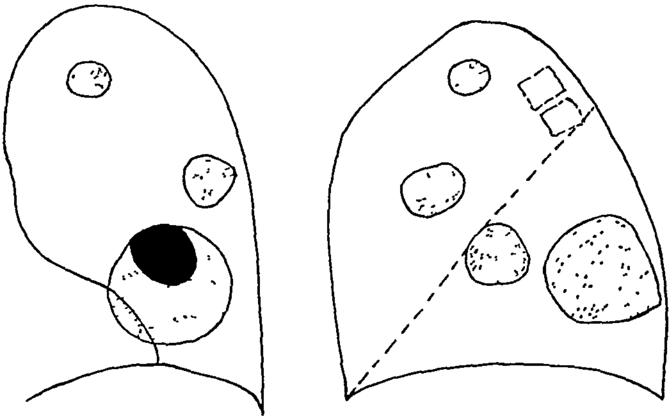

Fig. 2.—Schematic drawing of the localization of the processes as seen at exploratory thoracotomy.

Bronchoscopy showed nothing abnormal, and histologic examination of the bronchial secretion contained no cells ascribable to a malignant process. However, since there was still reason to suspect that the roentgenographic lung changes were malignant, exploratory left-sided thoracotomy was performed on Sept. 14, 1956.

No pleural changes were observed. However, a flat, well-defined and slightly nodular tumor, the size of a billiard ball, was palpated at the border between the apical and basal segments of the lower lobe and just below the visceral pleura. Adjacent to this lump was a similar tumor the size of a walnut, which extended to the oblique fissure. In adjacent parts of this apical segment of the lower lobe were smaller indurated foci. Two more walnut-sized tumors of the same nature were felt in the upper lobe. One was located in the upper lingual segment and the other, which was not demonstrable in the roentgenograms, was located in the anterior subsegment of the apical segment (Fig. 2).

All the tumors felt alike: they were firm to hard and surrounded by macroscopically normal lung tissue. The smaller of the two lower lobe tumors was enucleated for rapid histologic diagnosis. The microscopic picture was not suggestive of malignant tumor. The process was of homogeneous character and contained abundant amyloid deposits. On surface was finely nodular The cut surface was gray white to gray yellow and vitreous, but in some places granular with scattered, chalky spots In addition, diffusely outlined gray black foci, anything up to the size of confetti, were noted (Fig 4) The surrounding lung parenchyma was aerated rich in blood and the site of moderate anthracosis The

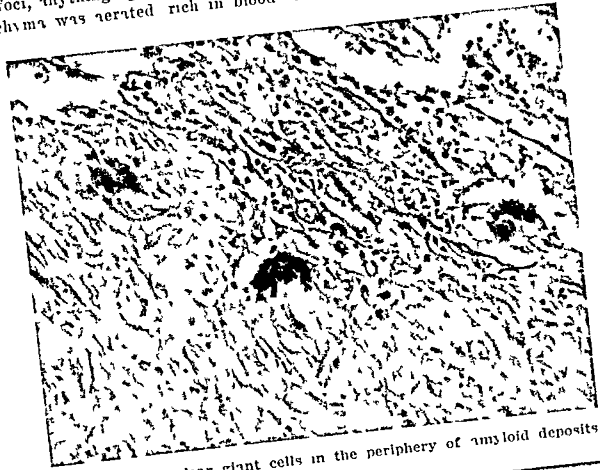

Fig 5 —Multinuclear giant cells in the periphery of amyloid deposits

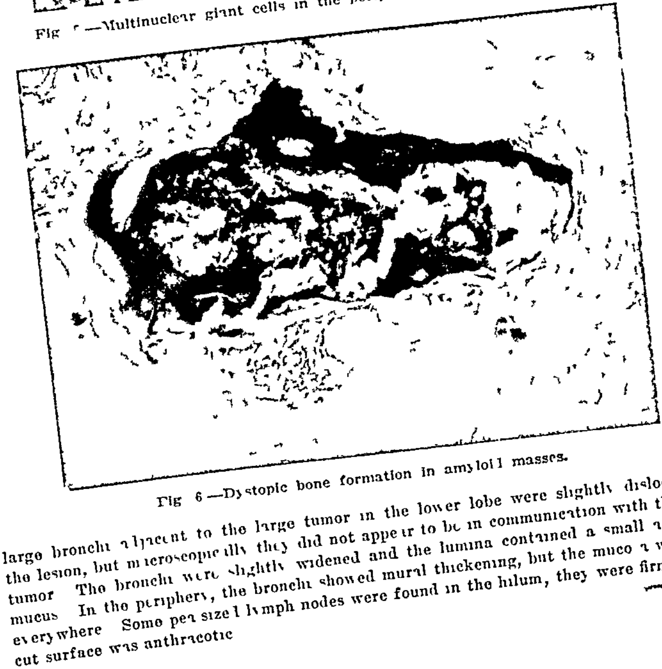

Fig 6 —Dystopic bone formation in amyloid masses.

large bronchi adjacent to the large tumor in the lower lobe were slightly dislocated by the lesion, but microscopically they did not appear to be in communication with the actual tumor The bronchi were slightly widened and the lumina contained a small amount of mucus In the periphery, the bronchi showed mural thickening, but the mucosa was intact everywhere Some pea sized lymph nodes were found in the hilum, they were firm, and the cut surface was anthracotic receipt of the pathologist's report, the operation was continued with removal of the lower lobe and enucleation of the two upper lobe lumps. Nowhere had the tumor involved the large bronchial branches.

The postoperative course was uneventful, and 14 days later the patient left the hospital (Sept. 29, 1956). During the first few weeks convalescence was smooth, and the patient had no respiratory tract symptoms. However, he fell acutely ill on Oct. 12, 1956, with a virus infection of influenza type. and 2 days later expectoration was abundant and sometimes sanguinolent. The patient was readmitted to the department of thoracic surgery on October 16. Roentgen examination of the lungs showed an intrapleural space partly filled with fluid and located at the earlier site of the enucleated tumor in the apical segment of the upper lobe. Intense antibiotic therapy controlled the fever, and the initially abundant expectoration ceased. Three weeks later, slight albuminuria and microscopic hematuria were noted. These signs were still persistent when the patient left the hospital in an otherwise good condition on Dec. 1, 1956. Since then the patient has had no respiratory tract symptoms (Fig. 3).

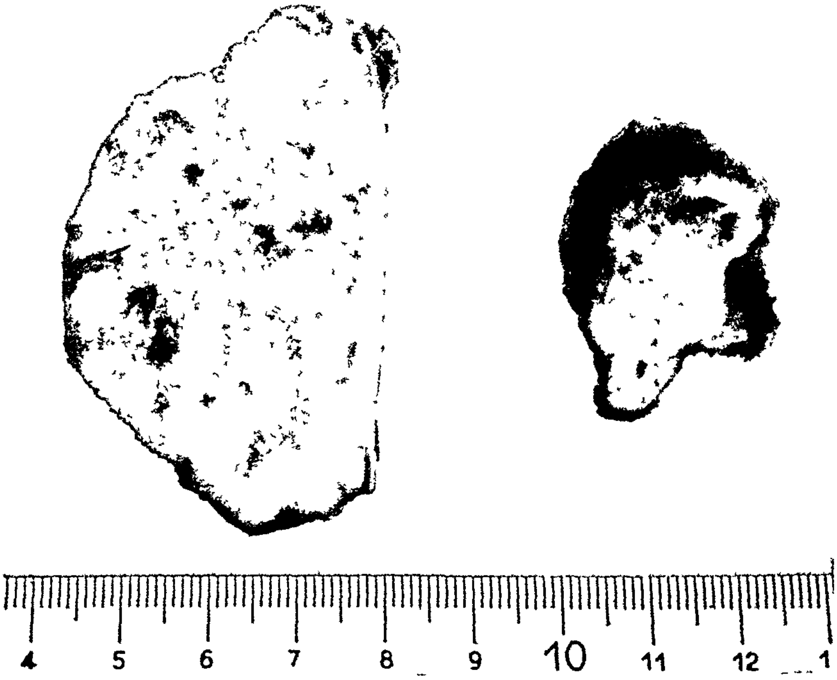

Fig. 4.—Gross specimens from the large tumor in the lower lobe and from one of the tumors in the upper lobe. The cut surfaces were waxlike with scattered ill-defined darker portions.

Supplementary Examinations.—Because of the histologic picture of the processes, the blood and urinary proteins were repeatedly determined, but without providing any support for a diagnosis of myeloma. Further roentgen examinations of the skeleton were also negative. Electrophoretic analysis of proteinbound blood carbohydrate showed normal values. The blood-forming bone marrow was normal. Further examination, because of persistent renal symptoms, suggested the possibility of glomerulonephritis. The patient is still under observation.

Macroscopic Examination.—The specimen consisted of the entire left lower lobe and 2 tumors from the upper lobe. The largest diameter of the tumor in the lower lobe was 6 cm. The other tumors were barely the size of walnuts. The tumors were well defined against the surrounding lung parenchyma, which was markedly compressed at several sites and the tumors were enucleated without difficulty. The tumors were firm or hard and the

In some of the large arteries, all of the layers of the wall contained deposits, and one large bronchus contained foci of hyaline substance in the mucosa, whose epithelium showed squamous epithelial metaplasia.

The deposits stained pink with hematoxylin and eosin. The intensity of the color varied markedly from one portion to another. In preparations stained with van Gieson stain, the deposits were orange and thin, red bands of connective tissue were seen between the coarse trabeculae. Of our most common routine amyloid staining methods, methyl violet often stained the trabeculae bright red, but the degree of metachromasia varied widely in different regions. Congo red stained the hyaline substance red in large regions, but often not strong enough to distinguish from connective tissue and the surrounding lung parenchyma. When stained with Lugol and iodine sulphuric acid, small regions were seen with an olive green hue, while the bulk of the masses did not show the color reaction typical of amyloid.

The tumors often showed foreign body giant cells with numerous irregularly arranged, rounded, or oval nuclei and homogeneous cytoplasm, which often contained substance which stained metachromatically with methyl violet. The giant cells often lay in the periphery of the deposits (Fig. 5), but were sometimes seen centrally in the tumors in and close to small slit shaped spaces around irregularly shaped chalky lamellae and bone trabeculae (Fig. 6). In addition, these regions contained abundant round cells, most of which were plasma cells. In the large tumor in the lower lobe, close to small epithelial lined spaces (mucus glands), were single regular cartilaginous islands (Fig. 7) probably representing rests of a completely changed bronchus.

In addition to the staining methods for amyloid, the histologic preparations were studied by elastic staining according to Weigert and silver impregnation according to Hortega as modified by King.[illegible] Frozen sections of the preparations were also stained with scharlach I. In sections stained for elastic tissue, the amyloid assumed a weak, brown red color. The amyloid masses often contained poorly outlined vessel structures with stratified, round, or oval formations with central endothelial lined spaces filled with red blood cells (Fig. 8). The lumen was markedly narrowed and the thickened vessel wall contained rests of concentrically arranged elastic lamellae. On silver impregnation, amyloid masses in large regions showed intense argyrophilia, while in other regions they did not take on any stain at all. The amyloid trabeculae often showed small, clear vacuoles which, when stained with scharlach I, took on a clear red color.

DISCUSSION

The case just described agrees with most of those on record and the changes observed fill the criteria usually accepted for a diagnosis of nodular amyloidosis: (1) the macroscopic appearance of the deposited substance and the presence of circumscript foci, (2) typical color reaction with amyloid staining methods but also poor agreement with different methods (Lubarsch[8]), which some authors (Glauser and others[7]) regard as suggestive of chemical differences in the amyloid deposit, and (3) lack of predisposing so called basal disease.

Whether the amyloid had been deposited also in organs other than the lung in the case under discussion can, of course, not be decided. No clinical symptoms were available to support such an assumption.

Further features regarded as characteristic of nodular amyloidosis are deposits of multinuclear giant cells and the occurrence of bone. Thus, as pointed out by Tsunoda and others,[13] giant cells occur almost regularly in localized amyloidosis but not in generalized amyloidosis. Tsunoda,[13] von Werdt,[14] and others claimed that the cells consist of phagocytic [illegible] of the conne

Histologic Examination.—The tumorlike deposits in the lung were built up of closely packed homogeneous trabeculae and laminae, poor in cells, between which were thin streaks of connective tissue with spindle-shaped cells. In large regions the tissue was loose and granular. Between the trabeculae were small deposits of carbon. Adjacent to and often radiating from the localized circumscript deposits were other deposits in alveolar septa and neighboring vessels and bronchi. The alveolar septa were thickened and the capillaries were markedly narrowed. The changes decreased toward the periphery.

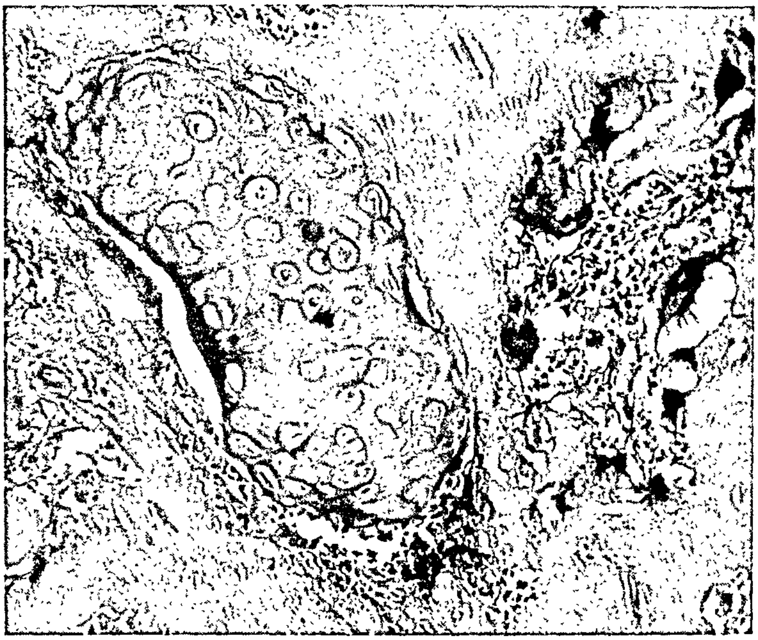

Fig. 7.—Deposit of multinuclear giant cells and round cells in slit-shaped spaces adjacent to a cartilaginous island in the middle of the large lower lobe tumor.

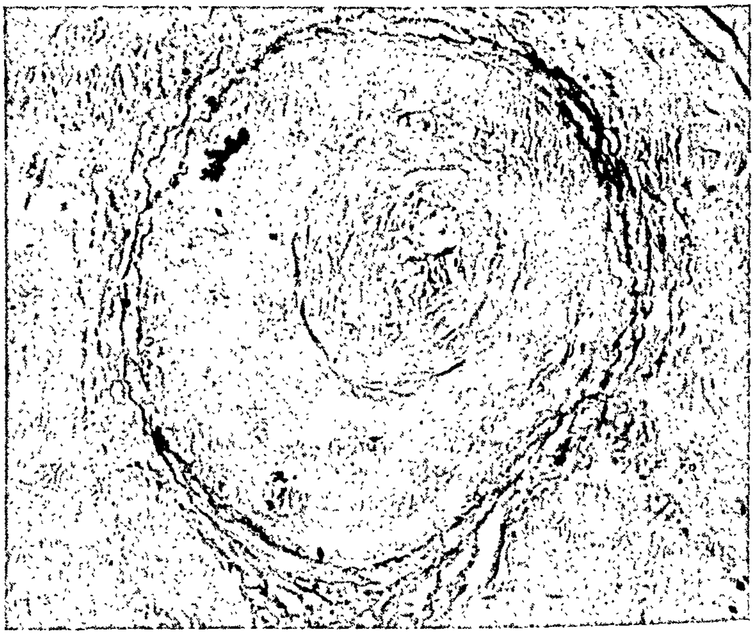

Fig. 8.—Artery with amyloid deposits in all layers of the wall and pronounced narrowing of the lumen.

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tissue or the vessel walls, while other authors (W. Fischer[1]) do not believe that giant cells have any phagocytic power but interpret them as a manifestation of a tissue reaction to a foreign substance. However, the fact that, as in the case under discussion, the cells often contain a substance staining like amyloid argues for the cells possessing phagocytic power. In experimental investigation on animals, Ljubimow[7] has shown that amyloid transplanted under the skin is absorbed by giant cells. Then, however, the amyloid absorbed by giant cells often loses its typical color reaction, a loss which has been accepted as a sign of chemical change in the absorbed amyloid.

Calcification and ossification in amyloid tumors have been observed in many cases on record. Small regular cartilaginous islands have also been reported. While the cartilage is probably derived from the bronchi, whose normal structure is obliterated by amyloid deposits, the spongy trabeculae are probably ascribable to an angious bone formation (Scheidegger[12]). The calcification is often accompanied by fatty degeneration of the tumors.

The occasionally abundant deposits of plasma cells in the amyloid masses merit comment. Diffuse or circumscript amyloid deposits are relatively common in multiple myeloma, and then usually with the typical localization of primary amyloidosis and in myeloma foci as well as in and around joints. In the present case, thorough, repeated clinical examinations failed to reveal any definite support for multiple myeloma, and the assumption that amyloid tumors in the present case are due to multiple myeloma and represent extramedullary myeloma foci appears less likely.

SUMMARY

A report is given of a case of multiple nodular amyloidosis of the lung in a man, aged 67. Roentgen examination revealed 3 of 4 rounded formations in the left lung. The lower lobe was removed and the tumors in the upper lobe were enucleated. Histologically, the tumors were built up of homogeneous masses, poor in cells and staining like amyloid. Abundant round cells, mainly plasma cells, were observed as well as multinuclear giant cells and, in some regions, deposits of bone and cartilage.

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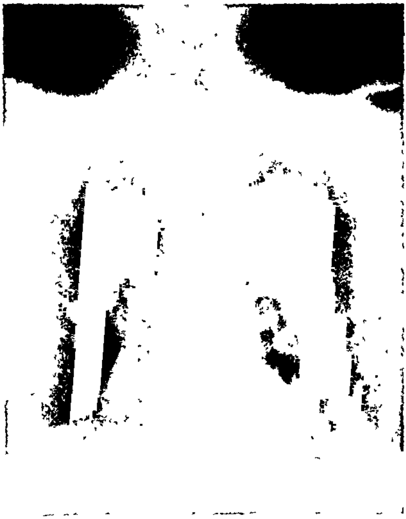

Fig. 1.

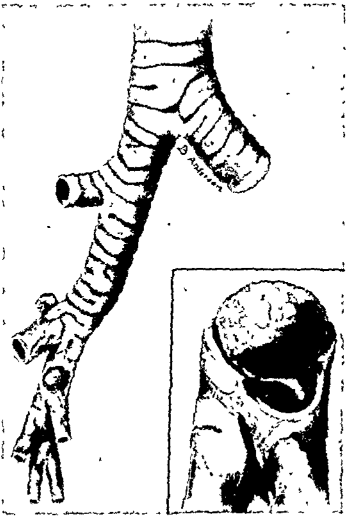

Fig. 2.

Fig. 1.—Case 1. Body section radiograph at 12 cm. which shows mass obstructing RLL bronchus and obstructive pneumonitis.

Fig. 2.—Case 1. Diagram which illustrates relationship of lipoma to RLL bronchus. Wrinkled surface of tumor is shown in inset.

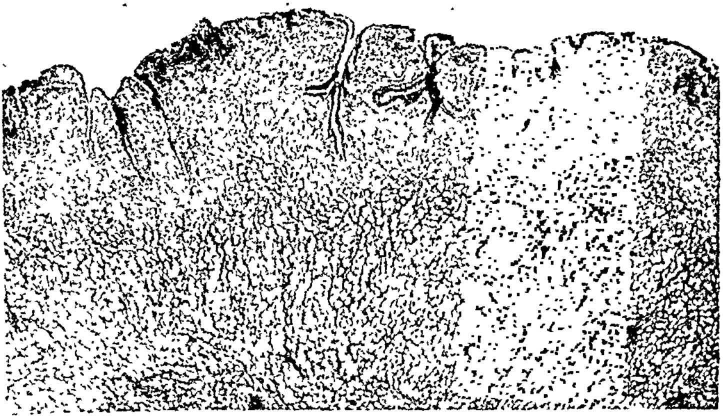

Fig. 3.—Case 1. Lipoma covered by wrinkled surface. Subepithelial tissues scarred and infiltrated by chronic inflammatory cells. (×25; reduced ¼.)

LIPOMAS OF THE BRONCHUS

A Presentation of Two Cases and an Analysis of the Literature

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BRONCHIAL lipomas, as well as other benign bronchial neoplasms, formerly pathologic curiosities, have become surgically remedial conditions as techniques for accurate diagnosis and successful removal have been developed.

Kernan,[1] in 1927, was the first to treat a bronchial lipoma by surgical means when he successfully removed one by bronchoscopy. Prior to this time three such lesions had been reported from autopsy material.[2-4] Since Kernan's report, however, 19 additional lipomas or lipofibromas have been treated by bronchoscopy,[5-15] bronchotomy,[16] or where irreparable lung damage was present, by lobectomy[17-19] or pneumonectomy,[20] while only 2 cases have been added from autopsy sources.[21, 22] Thus, the modern management of another disease process capable of destroying the host has developed from knowledge gained through post-mortem studies.

All benign endobronchial tumors fundamentally exert similar influences on the lungs; by partial to complete obstruction of the bronchial lumen, the egress of normal secretions from the bronchial mucosa is obstructed, and inflammation in and around the smaller bronchi and bronchioles occurs with pneumonitis, abscess formation, and/or bronchiectasis as the result.

CASE REPORTS

Case 1.—A 56-year-old white man was admitted to Baylor University Hospital because of a nonproductive, persistent cough of 10 months' duration. Ten months previously he had developed a "respiratory, flu-like infection." Two months before admission he became feverish and an x-ray examination of the chest was reported as showing fibrosis and pleuritis.

Posteroanterior, lateral, and laminographic examinations of the chest (Fig. 1) revealed pneumonitis and atelectasis of the basilar segments of the right lower lobe. A nodularity in the region of the hilus was suggestive of carcinoma.

Bronchoscopic examination demonstrated a rounded, smooth, pulsating, epithelized mass just beyond the superior segmental orifice of the right lower lobe which almost completely occluded the lumen of the basilar segmental bronchus. Biopsy of the pulsating tumor was considered inadvisable.

Thoracotomy by one of us (W. M. A.) revealed the basilar segments of the lower lobe to be markedly involved in suppurative pneumonitis with thick vascular adhesions extending

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measuring 2 by 1 cm. and projected proximally for a distanre of 3 cm. The consistency of the lesion was similar to yellow greasy fat. The upper lobe parenchyma was dense and fibrotic and the bronchi were dilated with mucopurulent material and had thickened walls. The middle and lower lobes were congested and somewhat more fibrous than normal.

On *microscopic examination* the tumor consisted of adult fat tissue covered by respiratory epithelium. All lobes showed chronic pneumonitis with fibrosis, particularly the upper lobe. There was a notable population of eosinophils throughout all lobes. The pathologic diagnosis was polypoid lipoma of the right upper lobe bronchus with bronchiectasis of the right upper lobe and fibrosis and chronic pneumonitis of the right upper, middle, and lower lobes. The patient has had no further difficulties in the 12 years since his operation.

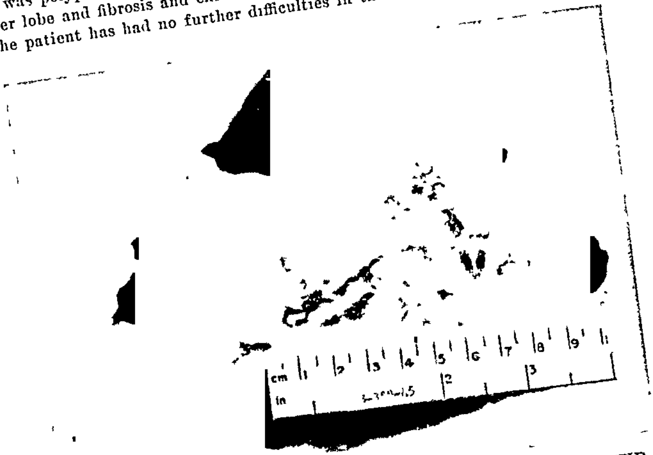

Fig 5.—Case 2. Polypoid lipoma projecting into right main bronchus (AFIP Acc. No. 138325-3)

COMMENT

The stimulus that induces fat which is normally present in the bronchus[19] to undergo neoplastic transformation is unrecognized. Bronchial lipomas most frequently affect individuals in the fifth decade, the average age being 51.1 years among the 20 cases that have been treated surgically, the youngest 29[13] and the oldest 64 years of age.[6] Eighty-eight per cent occurred in men. The duration of symptoms varied from 7 weeks[16] to 15 years[12] with an average duration of 3 years in 13 cases. The symptoms, signs, and radiologic findings were those of bronchial stenosis with accompanying pulmonary parenchymal disease. In several cases the radiographs were suggestive of bronchial carcinoma. The majority of the tumors could be visualized by bronchoscopy. The tumors arose from the bronchial tree in the following locations: trachea, 1; left main, 8; left upper lobe, 3; left lower lobe, 2; right main, 3; right upper lobe, 2; right lower lobe, 2. The composite dimensions of the tumors in the 12 cases treated surgically, where accurate measurements were given, were 2.5 by 1.3 cm., and the tumors and pulmonary changes reported in the present paper were representative of the group previously reported.

between the visceral and parietal pleura. The hilar and right middle and lower peribronchial lymph nodes were moderately enlarged, soft, and succulent. The upper and middle lobes appeared unremarkable. Accordingly, the right lower lobe was resected.

Pathologic examination: The specimen consisted of a right lower lobe which weighed 335 Gm. A pyriform tumor arose by a narrow pedicle from the wall of the basilar segment and projected proximally to occlude almost the main bronchus of the basilar segment (Fig. 2). The tumor measured 13 by 10 by 10 mm. while its stalk measured 3 mm. in length and 2 mm. in diameter. Its external surface was wrinkled and its interior was of a yellow-gray, solid, rubbery consistency.

The basilar segments of the lobe were atelectatic and nodular while the superior segment was of normal consistency.

Microscopic examination (Fig. 3) disclosed a relatively smooth, intact, respiratory epithelium covering the tumor with frequent epithelial invaginations. In some places the epithelium was only one cell in thickness, while in other places, squamous metaplasia had occurred. The interior was composed of adult fat cells supported by a scanty fibrovascular stroma. A zone of fibrous scar tissue separated the surface epithelium from the interior of the tumor. The pathologic diagnosis was fibrolipoma of the right lower lobe bronchus with chronic obstructive pneumonitis. The patient has been free of symptoms during the ensuing 16 months.

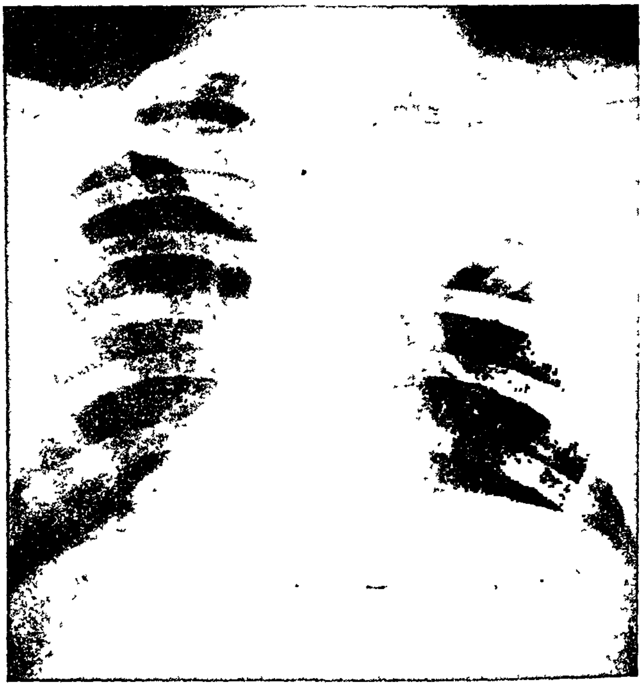

Fig. 4.—Case 2. Atelectasis of RUL which resulted from bronchial obstruction due to lipoma. (AFIP Acc. No. 138325-3.)

CASE 2.—A 32-year-old white man was admitted to the Brooke General Hospital, Fort Sam Houston, Texas, because of a cough of 21 months' duration, accompanied by a loss of 35 pounds of body weight. Pains in the back and right chest had been present for one year. X-ray studies revealed atelectasis of the right upper lobe (Fig. 4). As a biopsy obtained by bronchoscopy was suggestive of anaplastic carcinoma, a right pneumonectomy was performed by one of us (D. L. P.).

Pathologic examination: The pleural surfaces of all lobes were covered by thin fibrinous adhesions. At the origin of the right upper lobe bronchus a polypoid mass filled the lumen and projected into the right main bronchus (Fig. 5). It was attached by a base

EFFECT OF EXPERIMENTAL PULMONARY VALVULAR INSUFFICIENCY ON THE CIRCULATION

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BARGER, Roe, and Richardson[1] produced pulmonary valvular insufficiency in 3 dogs by means of damage to 1 to 3 cusps. These workers concluded that pulmonary insufficiency alone had little effect on mean right auricular pressure or work tolerance in the dog, even after months of strenuous exercise. Ellison, Brown, Hague, and Hamilton[2] studied 10 dogs who survived total pulmonary valvectomy for 1 to 14 months. None of these dogs presented evidence of heart failure. Right ventricular end-diastolic pressures, cardiac outputs, and heart volumes determined by x-ray studies remained normal. In an earlier paper, Fowler, Mannix, and Noble[3] found no increase of right ventricular diastolic pressure in 8 dogs studied 2 weeks to 6 months after removal of one or portions of 2 pulmonary valve cusps.

The purpose of the present study was to evaluate the long-term effect in dogs of more extensive pulmonary valve resection than was done in our earlier work.[3] Since our findings differ in some respects from those of Barger and co-workers[1] and those of Ellison and associates,[2] it is believed worthwhile to present them.

MATERIALS AND METHODS

Attempted total pulmonary valvectomy was done in 18 mongrel dogs under anesthesia with pentobarbital sodium, 30 mg. per kilogram. Right ventricular circulation was interrupted for 2 minutes or less, by occlusion of the superior vena cava, inferior vena cava, and coronary sinus. After the right ventricle had expelled most of its blood, the main pulmonary artery was clamped. The right ventricle was opened, and the valve cusps were excised under direct vision. The edges of the right ventricular incision were approximated by a Potts clamp, and the circulation was re-established. The operative wound was then closed with interrupted silk sutures.

Right atrial pressures were recorded through a cardiac catheter with the chest closed. Right ventricular and pulmonary arterial pressures were recorded simultaneously preoperatively and postoperatively through No. 19 needles with chest and pericardium open. Pressures were detected by Statham P23D transducers or Sanborn electromanometers. Pressures were recorded upon the Sanborn Poly-Viso electrocardiograph or the Electronics for Medicine Research Recorder.

In 8 animals, surviving 11½ to 18 months, further studies were made. At 4 to 6 months after operation, right heart pressures were measured through a

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SUMMARY

Two cases of bronchial lipoma are reported which brings to 20 the total number of cases in which surgery was performed. Five additional cases have been recorded from autopsy material.

Lipomas as well as other benign bronchial neoplasms are capable of causing irreparable pulmonary parenchymal damage and on occasion may result in destruction of the host. It is for these reasons that early diagnosis and surgical treatment are of great importance.

Permission obtained from the AFIP, Capt. W. M. Silliphant, Director, for the incorporation of data from Case 2 is gratefully acknowledged.

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Teleroentgenograms of the chest, taken just prior to sacrifice, showed definite evidence of cardiac enlargement in only 2 animals, Dogs 14B and 20B. In each of these, the pericardium could not be closed at the time of the origin
operation because of right ventricular dilation. The anteroposterior and righ
lateral teleroentgenograms of Dog 20B are shown in Figs 3 and 4. The pr
operative films are on the left in each figure.

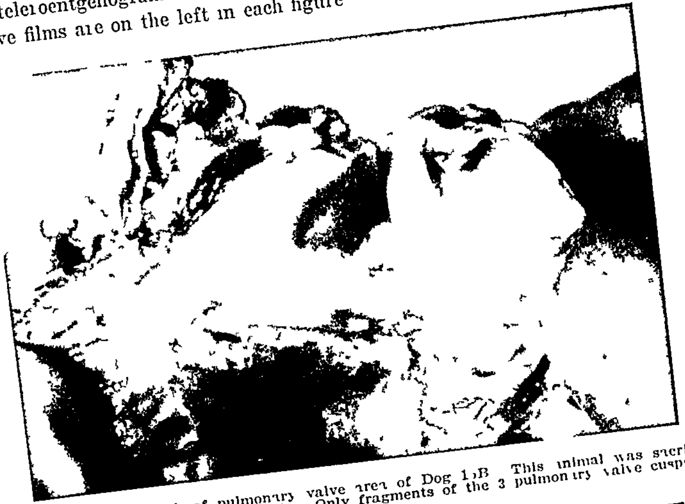


Fig 1.—Photograph of pulmonary valve area of Dog 1[illegible]B. This animal was sacrificed 11 months and 3 weeks postoperatively. Only fragments of the 3 pulmonary valve cusps are remaining.

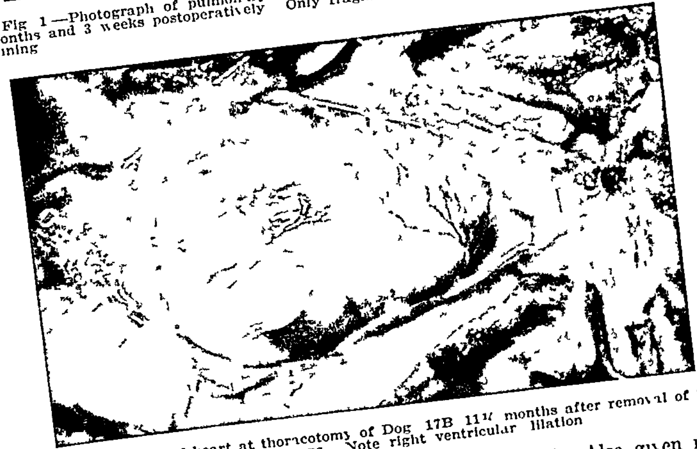


Fig 2.—Photograph of heart at thoracotomy of Dog 17B 11½ months after removal of 3 pulmonary valve cusps. Note right ventricular dilation.

The results of pressure studies are given in Table I. Also given is the
number of pulmonary valve cusps removed as establishe autopsy. PA and
RV pressures were obtained satisfactorily in 11 ately bef

cardiac catheter, and cardiac outputs were measured by the direct Fick principle. At 11½ to 18 months after operation, the animals were sacrificed. Posteroanterior and right lateral teleroentgenograms of the chest were made preoperatively, at 4 to 6 months after operation, and prior to final sacrifice. Twelve lead electrocardiograms were made preoperatively, at 4 to 6 months after operation, and prior to final sacrifice.

Just prior to final sacrifice, other observations were made. Right atrial pressure and direct Fick cardiac outputs were determined in the anesthetized, closed chest animal. Electrical stimulation of the four extremities was then begun at a frequency of 284 per minute, with a pulse duration of 0.022 sec. and a current of 50 Ma. Stimulation was carried out for 10 minutes, with direct Fick cardiac output measurement during the last 3 minutes. After a 30-minute rest, the direct Fick cardiac output was again measured. Electrical stimulation and measurement of cardiac output were then repeated as before. The animal's chest was then opened under positive pressure respiration. Simultaneous measurement of right ventricular and pulmonary arterial pressure was made, and photographs of the heart were taken. The animal was then sacrificed. Liver biopsy was taken and the peritoneal cavity inspected for evidence of ascites. The heart was removed, weighed, and the pulmonary valve area was examined. The heart was then fixed in formalin for 2 to 4 weeks. Right and left ventricular weights were then determined by the method of Herrmann.[4]

RESULTS

Satisfactory pulmonary valvectomy was performed in 18 animals. Of these, 8 failed to survive the operation or immediate postoperative period because of excess blood loss, pneumothorax, or ventricular fibrillation associated with right ventricular dilation after valvectomy. One animal died one month postoperatively of extensive bronchopneumonia. The right ventricle and right atrium of the animal were dilated at autopsy. Another animal died 4 months after operation following a cineangiocardiogram, which demonstrated regurgitation of dye from pulmonary artery to right ventricle. Thus, 8 animals remained. These were sacrificed after 11½ to 18 months' observation, during which time they were allowed to exercise frequently and displayed no limitation of activity. Two animals had litters of 9 puppies each without apparent difficulty.

Autopsies were done in 10 animals surviving the operation. All 3 valve cusps had been removed in 5 animals; 2 cusps had been removed in 4; one cusp had been removed in one. Fig. 1 shows the pulmonary valve area of Dog 15B; only fragments of valve cusps are remaining. This animal was sacrificed 11 months and 3 weeks postoperatively.

At the time of the original operation, right ventricular and right atrial dilation occurred in each animal following pulmonary valvectomy. In 3 of the 18 animals, the right ventricle was dilated to an extent which rendered closure of the pericardium impossible. At the time of final thoracotomy prior to sacrifice, the right ventricle appeared dilated in each animal, but only after the pericardium was opened. Fig. 2 shows right ventricular dilation in a photograph of the heart of Dog 17B taken during thoracotomy just prior to sacrifice after survival of 11½ months. All 3 pulmonary cusps had been removed.

diastolic pressure rose in seven instances. Where measured, right atrial pressure rose 0.5 to 1.5 mm Hg immediately after operation. At the time of final study, one animal expired before pressures could be measured. At this time, right ventricular diastolic pressures were, in general, lower than immediately postoperatively. Fig. 5 shows simultaneous right ventricular and pulmonary arterial pressures of Dog 20B, showing RV pressure of 23/1.5 and PA pressure of 23/4 mm. Hg just prior to sacrifice, 11½ months after total pulmonary valvectomy. Right atrial pressures, however, were approximately as high or higher in three instances where comparisons could be made.

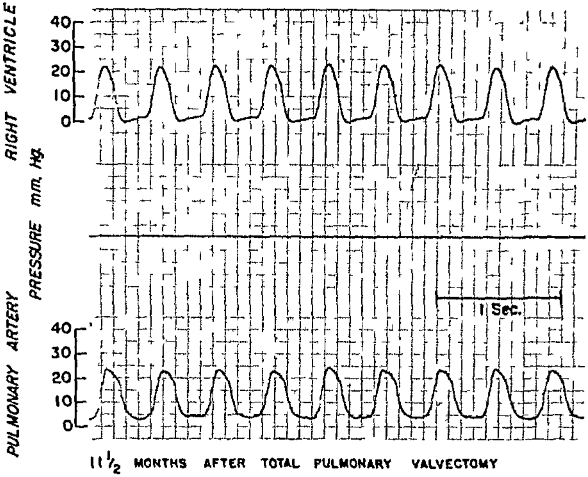
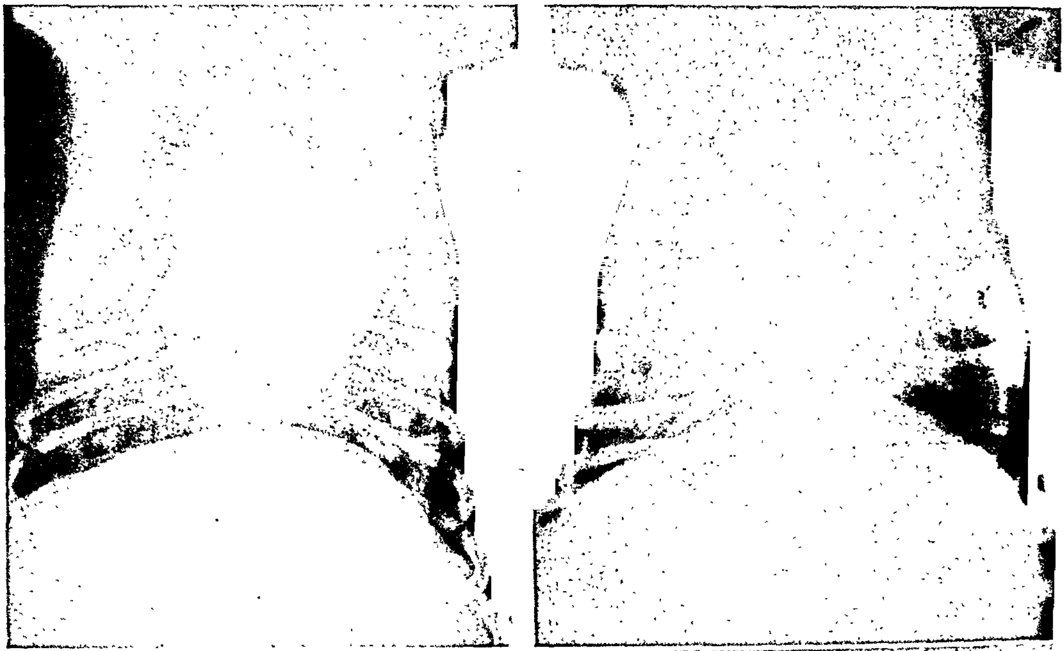


Fig. 5.—Simultaneous right ventricular and pulmonary arterial pressure record of Dog 20B. Pressures were taken 11½ months after total pulmonary valvectomy. RV pressure is 23/1.5 mm. Hg and PA pressure is 23/4 mm. Hg. PA pressure was 24/14 mm. Hg preoperatively.

Cardiac outputs were measured 4 to 6 months after operation in the survivors (Table II). The mean of cardiac outputs at this time was 199 ± 21 (S.E.) c.c./Kg./min. This value was compared with the mean of cardiac outputs in a control series of 52 normal anesthetized dogs studied in this laboratory, whose values were 161 ± 7.7 c.c./Kg./min. The difference was not significant statistically, $p > 0.05$. At the time of final study, the mean of cardiac outputs was 111 ± 14.9 c.c./Kg./min. This value was significantly lower than in the control group, $p < 0.05$, and was significantly lower than in the first postoperative study, $p < 0.05$. The first electrical stimulation increased mean cardiac output 178 ± 29 c.c./Kg./min., $p < 0.01$. Mean cardiac output fell to 131 c.c./Kg./min. 30 minutes after exercise and rose to 285 c.c./Kg./min. during electrical stimulation. The mean increase of 154 ± 33 c.c./Kg./min. was significant, $p < 0.01$.

immediately following valvectomy. It may be seen that PA diastolic pressure fell in each instance; control values were 4 to 19 mm. Hg; a fall of 3 to 14 mm. Hg to values of 0 to 8 mm. Hg occurred following valvectomy. Right ventricular

Fig. 3.



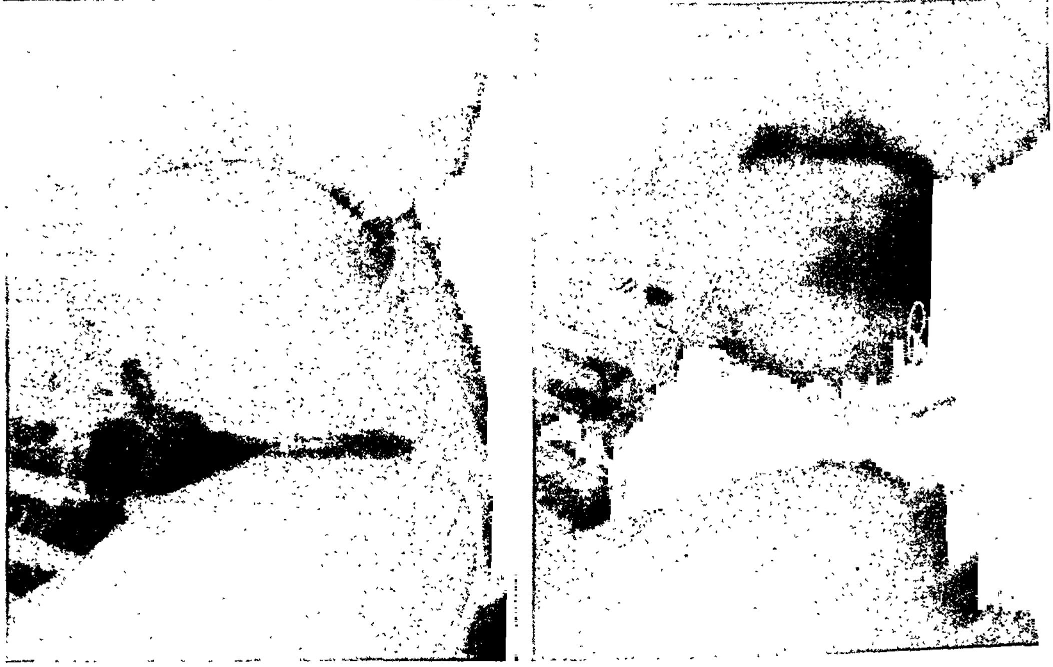


Fig. 4.

Fig. 3.—Anteroposterior teleroentgenograms of the chest in Dog 20B. *On the left is reproduced the preoperative film. On the right is the roentgenogram taken 11½ months after removal of all 3 pulmonary valve cusps, showing increase in cardiac size. The pericardium could not be closed postoperatively in this dog.*

Fig. 4.—Right lateral teleroentgenograms of Dog 20B which shows increase in cardiac size 11½ months after total pulmonary valvectomy. *The preoperative film is on the left.*

DISCUSSION

There are several indications in the study described above that severe pulmonary valvular insufficiency is not a benign lesion in its effect upon the circulation. Although our 8 animals which survived approximately one year were still apparently well, there were evidences of right heart dilation at the time of valvectomy and at the time of final sacrifice in each animal. In 3 of 10 surviving animals, right ventricular dilation at the time of valvectomy was so great as to preclude pericardial closure. In addition, the mean resting cardiac output, although normal 4 to 6 months postoperatively, was below normal approximately one year postoperatively. In this respect, our conclusions differ from those of Barger and co-workers,[1] and from those of Ellison and associates.[2] Also, we failed to find a systolic pressure gradient between right ventricle and pulmonary artery in the presence of pulmonary insufficiency as described by Ellison and co-workers.[2]

In man, primary pulmonary valve disease as a cause of pulmonary insufficiency is uncommon, and it is thus difficult to gain a clinical impression of the importance of this disturbance. McGuire and McNamara[5] described patients with organic pulmonary insufficiency; since other valves were involved in each of their patients, it is difficult to know what effect was produced by the pulmonary insufficiency. Kohout and Katz[6] reported a case in which a clinical diagnosis of pulmonary insufficiency was made This patient showed evidence of right heart enlargement and had had heart failure. However, ventricular septal defect was probably present in addition to the lesion of the pulmonary valve Campeau, Ruble, and Cooksey[7] reported a case of congenital absence of the pulmonary valve. Although their patient had congestive heart failure, it is impossible to ascertain the relative importance of the pulmonary valve lesion, since the patient had also a ventricular septal defect and a single coronary artery with myocardial infarction. Ford, Hellerstein, Wood, and Kelly[8] reported a case of pulmonary insufficiency associated with a congenital bicuspid pulmonary valve Their patient, a woman of 43, developed right heart failure. She had also inactive pulmonary tuberculosis. These authors concluded from a review of the literature that congenital pulmonary incompetence due to bicuspid valve does not produce congestive heart failure unless other cardiac or pulmonary disease is present Kjellberg, Mannheimer, Rudhe, and Jonsson[9] described a boy of 7 years in whom a clinical diagnosis of isolated pulmonary valvular insufficiency was made. This patient showed no evidence of right ventricular enlargement

The effect of pulmonary valvular insufficiency upon the circulation is of importance, however, because of its occurrence as a complication of incision or partial excision of the congenitally stenotic pulmonary valve. Blount, McCord, Mueller, and Swan[10] stated that pulmonary insufficiency complicating the operation for pulmonary stenosis in 5 patients had produced no significant hemodynamic alterations, although they noted an increase in heart size postoperatively and thought that further observation was necessary. However, we have recently observed the development of right heart dilation in a boy of 6 years,

TABLE I. RIGHT HEART PRESSURES, MM. HG, BEFORE AND AFTER PULMONARY VALVECTOMY

DOG NO.	CUSPS REMOVED	INITIAL STUDY						FINAL STUDY		
		BEFORE			AFTER					
		PA	RV	RA	PA	RV	RA	PA	RV	RA
1B	1	20/12	23/1.6		23/7	18/6		28/9	27/1.5	4.3
8B	2	9/4	13/1.2		22/1	16/1		25/4	20/0	7.0
9B	2	21/10	22/0		8/5	10/4		21/4.5	18/0	4.8
10B	–	18/11	18/2		16/1	23/0.5				
12B	3	21/11	19/2		23/0	36/1				
14B	2	27/19	31/0		26/5	25/2		20/5*	45/2.5*	3.8
15B	3	24/14	23/4	4.5	29/8	40/6	5.8	27/4	27/6	5.5
16B	3	20/13	20/4	3.5	30/5	28/4	4.5			
17B	3	14/7	15/2	2.8	25/4	26/4	3.8	25/6	20/1	3.8
18B	2	18/12	14/0.6	3.0	33/5	38/3	4.5			
20B	3	24/14	24/1	2.5	26/6	19/5	3.0	23/4	23/1.5	6.5

PA and RV pressures were measured with chest open.

RA pressures were measured with closed chest; 5 cm. up from table is zero for RA pressures.

*Catheter pressures.

TABLE II. CARDIAC OUTPUTS IN C.C./KG./MIN. IN 8 DOGS SURVIVING PULMONARY VALVECTOMY 11½ TO 18 MONTHS

DOG NO.	4 TO 6 MONTHS AFTER OPERATION	11½ TO 18 MONTHS AFTER OPERATION			
	REST	REST	1ST ELECTRICAL STIMULATION	2ND REST	2ND ELECTRICAL STIMULATION
1B	180	102	157	120	179
8B	117	67	344	83	396
9B	238	133	281	117	259
14B	302	52	299	136	198
15B	178	127	325	130	304
17B	249	138	348	195	392
18B	175	–	–	–	–
20B	153	160	274	137	265
Mean ± S.E.	199 ± 21	111 ± 14.9	290 ± 24.7	131 ± 11.7	285 ± 32.3

None of the sacrificed animals had evidence of ascites at autopsy. Appreciable weight gains (up to 5 Kg.) were recorded during the year period of observation, but these were presumably due to improved nutrition.

Twelve lead electrocardiograms taken preoperatively and at restudy in 5 animals showed no evidence of right ventricular hypertrophy.

The heart weight/body weight ratio was slightly above the upper normal of 0.00994 given by Herrmann[4] in 2 animals in which values of 0.0105 and 0.0100 were obtained; however, right ventricular/body weight ratios were each within the normal ranges of 0.00178 to 0.00400 given by Herrmann.

Liver biopsy showed slight hepatic cell degeneration and early fibrosis in the central vein areas in only 2 animals, Dogs 15B and 17B.

PULMONARY FUNCTION BEFORE AND AFTER PULMONARY RESECTION IN TUBERCULOUS PATIENTS

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NUMEROUS reports in the medical literature indicate the interest that has centered on the reduction of pulmonary function accompanying resection of residual pulmonary foci following medical therapy of tuberculosis. Few studies have been reported, however, of groups of patients who had undergone resection without some concomitant space-filling procedure.

Loss of pulmonary function in patients with chronic pulmonary disease has been considered proportional to the extent of involvement of one or both lungs. Clinical and roentgenographic studies have not always provided a reliable index of the functional pulmonary capacity.[1, 2] Consequently, most studies of the effect of disease and of various types of therapy on pulmonary function have included preoperative and postoperative spirometric methods.[3-22] Bronchospirometry has also been employed when available and when unilateral variations have been anticipated.

There has been some controversy regarding the relation of loss of function to the number of segments resected. Van der Drift[4] and Limburg[5] indicated that pulmonary function is reduced less after segmental resection without complications than after lobectomy in patients with tuberculosis. They further stated that in larger unselected groups the reduction of function was similar whether segmental resection or lobectomy was carried out. Limburg suggested that this lack of difference might be caused by the greater incidence of complications among patients having segmental pulmonary resection. Taylor and associates[3] stated that loss of function following uncomplicated operations in a group of men with tuberculosis appeared to be unrelated to the increasing volume or number of segments resected, if less than a pneumonectomy. This, they thought, suggested the predominant influence of more constant factors.

However, Smith and associates,[7] in studying a group of young adults, mostly men, with bronchiectasis, stated that operation was found to produce further ventilatory loss proportional to the extent of the resection. They also

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From the Mayo Clinic and the Mayo Foundation, Rochester, Minn., and the Mineral Springs Sanatorium, Cannon Falls, Minn

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who had clinically and by right heart catheterization, only infundibular and valvular pulmonary stenosis. Following operation, which consisted of partial infundibular resection and incision of the pulmonary valve, the patient developed the murmur of pulmonary insufficiency and roentgen evidence of right heart dilation and abnormal peripheral pulmonary arterial pulsation. This was in evidence 3 months postoperatively. Cardiac enlargement of the same degree was still present 8 months postoperatively. One cannot be certain that this patient did not have some additional lesion, such as a small ventricular septal defect.

SUMMARY

Severe pulmonary valvular insufficiency was produced in 18 dogs, each of which developed right ventricular dilation postoperatively. In 3 animals, right ventricular dilation was so extensive that the pericardium could not be closed. Ten dogs survived the immediate postoperative period, and 8 dogs survived 11½ to 18 months and were sacrificed. There was no evidence of right heart failure nor of limitation of activity in the surviving dogs. Pulmonary artery and right ventricular pressure records showed evidence of severe pulmonary insufficiency. The mean of direct Fick cardiac outputs was normal 4 to 6 months after valvectomy and was below normal 11½ to 18 months after operation, but could still be increased by electrical stimulation of the extremities. At thoracotomy, after opening the pericardium, the right ventricle appeared dilated in each animal 11½ to 18 months postoperatively. Two animals of 8 long-term survivors showed evidence of cardiac dilation on teleroentgenograms. Two animals showed evidence of hepatic congestion on liver biopsy 11½ to 18 months after operation. It is concluded that severe pulmonary insufficiency in the dog is not innocuous even as an isolated lesion.

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saturation was determined by a recording "double-scale" ear oximeter.[26] Bronchospirometry was performed with modified Carlen's catheters in the supine position in the usual manner[27] for 20 patients.

Control measurements of lung volumes and maximal breathing capacity were obtained on 10 healthy adults (5 men and 5 women), repeated after periods of 2 weeks to 4 months.

RESULTS

Preoperative Pulmonary Volumes and Maximal Breathing Capacity.—Table I and Fig. 1 present the values obtained on healthy subjects and the preoperative values for the patients, classified in relation to the number of segments ultimately resected. The ages in the groups of patients did not differ greatly.

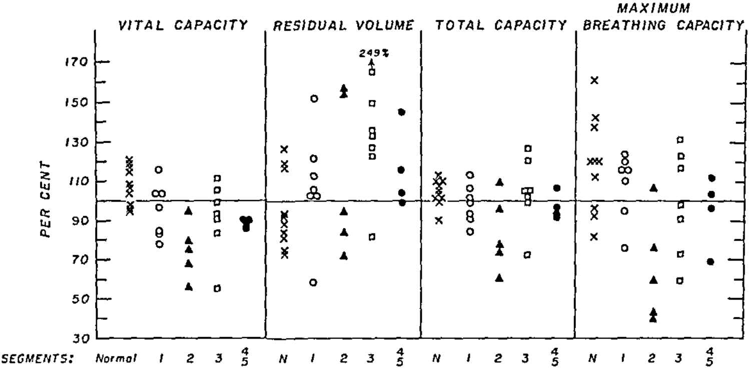

Fig 1.—Preoperative values in tuberculous patients. Per cent of predicted normal

TABLE I. AVERAGE VALUES OF PREOPERATIVE PULMONARY FUNCTION IN PER CENT OF PREDICTED NORMAL

SEGMENTS RESECTED LATER	SUBJECTS	MEAN AGE (YEARS)	VITAL CAPACITY	RESIDUAL VOLUME	TOTAL CAPACITY	MAXIMAL BREATHING CAPACITY
0 (Normals)	10	27	108	95	105	119
1	7	31	95	108	98	109
2*	5	43	74	112	83	63
3	7	43	91	142	105	99
4-5	4	41	90	116	98	95

*Low values attributed to significant bilateral disease in 3 of 5 patients.

The pulmonary volumes observed in the normal subjects agreed satisfactorily with predicted values, although the estimated maximal breathing capacities were usually somewhat smaller than observed.

Within each of the four groups of patients, there was considerable variation in the values for lung volumes and maximal breathing capacity, but the mean values were near normal except in the "two-segment" group, which had lower average values for vital, total, and maximal breathing capacities than predicted.

reported that a relatively uniform ventilatory loss which followed excision of little or no pulmonary tissue in 11 tuberculous men was related to a "thoracotomy effect" or change in the thoracic wall.

Several authors[4, 5, 8, 12] have pointed out that the amount of functioning lung removed determines the degree of impairment of function, and have stressed the importance of preserving as much normally functioning pulmonary parenchyma as possible in various chronic conditions.

The study reported here was undertaken to provide additional evidence on the effects of unilateral resection for tuberculosis on pulmonary function.

MATERIAL AND METHODS

Twelve men and 11 women who were patients at the Mineral Springs Sanatorium, Cannon Falls, Minnesota, were studied. Their ages varied from 21 to 62 years (average 39 years). Preoperative therapy for pulmonary tuberculosis consisted of modified rest in bed and chemotherapy with two or more drugs for an average period of 8 months. Resection was limited to bronchopulmonary segments containing residual areas of disease considered to be a threat to the patient's future. Segmental resection was done in preference to lobectomy if salvageable segments were found in the diseased lobes. No patient in this study had less than one segment removed. If a wedge resection of a small nodule was done in addition to removal of one or more segments, this was not counted in classifying a patient according to the number of segments removed. No space-filling procedures were done. Each patient's progress was followed by clinical examination, cultures of sputum or fasting gastric contents, and roentgenographic studies, including tomograms. Only patients without postoperative complications were included in this study. Patients who required somewhat prolonged pleural suction for pulmonary re-expansion, bronchoscopy for aspiration of temporarily retained secretions, or thoracentesis for effusion were not considered to have significant complications.

Pulmonary function studies were performed at the Mayo Clinic, usually within 2 weeks before operation. They were repeated 2 to 6 months after operation, usually 1 or 2 weeks prior to dismissal from the Sanatorium. Spirograms and total lung volume and its subdivisions were determined with a spirometer of Benedict-Roth type and the open circuit nitrogen elimination method for residual volume[23] modified as follows: Oxygen was inspired from one Neoprene bag of about 150 L. capacity, and expired gas was collected in another. Both bags were enclosed in a box, also connected to a monitor spirometer providing automatic correction for "switch-in" error and a continuous recording of respiratory excursions. The nitrogen concentration of gases near the mouthpiece was recorded continuously with a nitrogen meter and a photokymograph both to aid in detection of inspiratory leaks and to record the maximal concentration of nitrogen of the alveolar samples delivered after 7 minutes of oxygen inhalation, hereafter called the "nitrogen washout index." Normal values for lung volumes were estimated with the formula of Bateman[24] and for maximal breathing capacity with that of Baldwin and associates.[25] Arterial oxygen on The sum of the vital capacities of the two lungs recorded during broncho spirometry in the supine position was somewhat less than the total vital capacity measured in the sitting position about an hour before bronchospirometry Therefore, the vital capacity of each lung was multiplied by the ratio of standard vital capacity to total bronchospirometric vital capacity for each of the preoperative and postoperative measurements Thus, the postoperative unilateral reductions could be compared with the total reductions In 13 of 20 patients, the unilateral loss was calculated to be between 75 per cent and 125 per cent of the total loss, indicating a reasonable accounting for the total loss In 2 patients, the unilateral loss was more than 125 per cent of the total loss, indicating that the contralateral lung had increased its vital capacity In 5 patients, the unilateral loss was less than 75 per cent of the total loss, indicating some contralateral impairment after operation

TABLE II POSTOPERATIVE PULMONARY FUNCTION
(AVERAGE EXPRESSED IN PER CENT OF PREOPERATIVE VALUES)

SEGMENTS INVOLVED	SUBJECTS	VITAL CAPACITY	RESIDUAL VOLUME	TOTAL CAPACITY	FUNCTIONAL RESIDUAL CAPACITY	MAXIMAL BREATHING CAPACITY
0*	10	100	101	100	100	115
1	7	89	106	94	101	105
2	5	81	92	86	92	134
3	7	88	79	84	88	92
4 5	4	83	80	83	81	95

*Tests repeated in 2 to 16 weeks on normal subjects

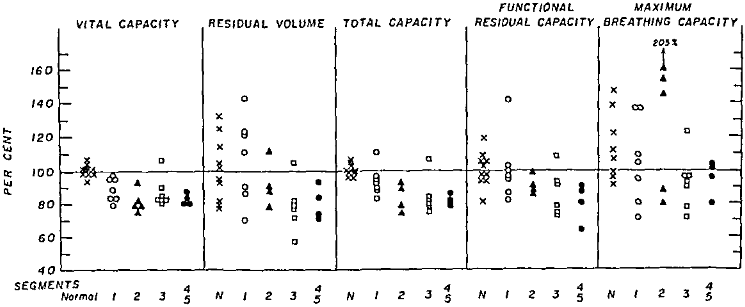

Fig 2—Postoperative values in tuberculous patients Per cent of preoperative values

With a few exceptions, reductions of oxygen uptake and respiratory minute volume of the lung operated on were found The magnitude of reductions was not closely related to the number of segments removed The amount of reduction in the percentage of the total minute volume contributed by individual lungs was more closely related to the amount of reduction of vital capacity than to that of oxygen uptake (Fig 4)

OTHER STUDIES

Postoperatively, arterial oxygen saturation was normal in each of the 21 patients measured at rest and in the 15 patients measured during exercise Of

This was caused by low values obtained on 3 of the 5 subjects who had had severe contralateral disease treated many years previously by pneumothorax.

Although the contralateral lungs in the 3 patients showed only minor roentgenographic abnormalities at the time of operation, it was apparent that contralateral disease had been extensive on the basis of the history, the previous thoracic roentgenograms, and preoperative bronchospirometric data which showed that the lung to be operated on was contributing a greater portion of the total function than the contralateral lung. The one patient in the three-segment group with markedly reduced values of vital, total, and maximal breathing capacity had undergone a contralateral five-rib thoracoplasty 6 years previously.

The size of the residual volume was related to age. Increased residual volumes, amounting to more than 125 per cent of the absolute value predicted for young adults, and also to more than 35 per cent of the total capacity, were found in 4 of the 6 patients more than 50 years of age and in only 2 of 17 patients less than 50 years of age.

The arterial oxygen saturation was normal (95 to 98 per cent) in all patients tested, both at rest (20 patients) and during several minutes of step-test exercise (12 patients). In only 4 of 23 patients was the pulmonary nitrogen washout index abnormal ($>$2.5 per cent), indicating the presence of retarded alveolar ventilation of some part of the lungs. These 4 men did not show the absolute and relative increases of residual volume that are usually associated with abnormal nitrogen indexes in chronic diffuse obstructive emphysema.

Postoperative Pulmonary Volumes and Maximal Breathing Capacity.—Table II and Fig. 2 show that, in control normal subjects, average pulmonary volumes were unchanged within 2 weeks to 4 months. Vital capacity and total capacity varied only within 94 to 107 per cent of the initial test.

In the patients, the postoperative value for vital capacity was less than the preoperative value in 22 of 23 cases, and the average reduction was similar in all 4 groups. In contrast, the residual volume and functional residual capacity were not consistently changed in patients having one or two segments removed, but were reduced in 9 of 10 patients in the three and four to five segment groups.

The total capacity was reduced in all groups, but not in a close relation to the number of segments resected. Postoperative values for maximal breathing capacity varied considerably from the preoperative value in many patients, but in no case were they reduced more than 30 per cent. In all groups, the average preoperative and postoperative values were similar for maximal breathing capacity and for the percentage of the "timed" or rapid vital capacity expired in one second (average 80 per cent). The average value of maximal midexpiratory flow[28] was 2.2 L. per second before and 1.9 L. per second after operation.

Bronchospirometric values are shown in Table III and Fig. 3. In 18 of 20 cases, the percentage of the total vital capacity contributed by the lung operated on was reduced postoperatively. An attempt was made to determine whether the postoperative reduction of total vital capacity could be accounted for entirely by the reduction in the vital capacity of the lung that was operated the 4 patients who had abnormal nitrogen washout indexes preoperatively, 2 remained abnormal, but 2 had normal indexes postoperatively, suggesting more uniform alveolar ventilation. After operation, 3 other patients had abnormal indexes. Estimates of the ratio of alveolar ventilation rate to functional residual capacity suggested that in one case the abnormal index probably indicated less uniform alveolar ventilation, and in another that the abnormality of alveolar ventilation had existed preoperatively but had been masked by an increased rate of ventilation.

EFFECT OF SEX AND AGE

The postoperative changes described in preceding paragraphs were of similar magnitude in both sexes and in patients more than or less than 40 years of age.

MULTIPLE RESECTIONS

Three patients underwent a second contralateral resection of one or two segments and were again tested 2½ to 4½ months later. In all, the maximal breathing capacity was unchanged after the second operation, the lung volumes (vital capacity, and so forth) were unchanged in one patient, but were further reduced by 5 to 10 per cent in 2 patients. A reduced contribution by the lung undergoing the second operation was observed in all cases, amounting to 5 to 15 per cent of the total function. Arterial oxygen saturations were normal at rest and during exercise in all patients.

COMMENT

The changes noted after operation in this study were similar to those of other reports. On examination, several months after resection of only one segment, or even after local enucleation of tuberculomas,[7] reductions of about 10 per cent in vital capacity are observed consistently,[3, 4, 15, 21, 22] with similar reductions of total capacity.[7, 21, 22] Reductions of residual volume or functional residual capacity are small and less consistent.[7, 22] Maximal breathing capacity and the one-second percentage of the rapid vital capacity are essentially unchanged. Resection of one segment causes the percentage of total ventilation, oxygen uptake, and vital capacity contributed by the side operated on to decrease about 5 percentage points, although the reductions may occasionally be as great as 20 per cent.[3, 4] Our data and those of Hirdes[21, 22] indicate that the loss of vital capacity on the side operated on largely accounts for the loss of total vital capacity.

After resection of two to five segments, the reduction in vital capacity,[3, 4, 23] residual volume, and total capacity[21, 22] is not much greater than after resection of one segment. The absence of a clear relationship between the number of segments removed and the volumetric reductions has been stressed by most authors. However, a trend of this type is apparent in our results and those of others.[3, 21]

Whereas the maximal breathing capacity is not consistently altered after resection of one segment, it is usually reduced 10 to 15 per cent after resection of four to five segments.[3, 4, 23, 22]

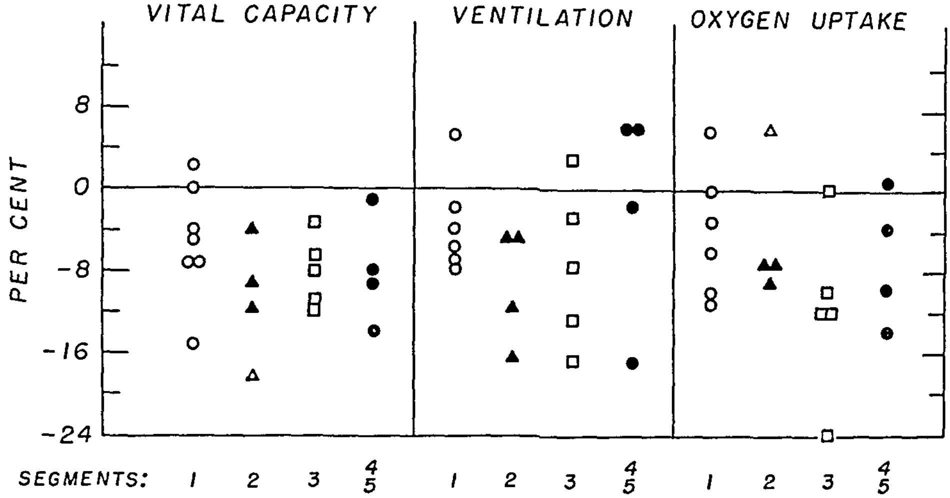

Fig. 3.—Change in lung operated on in per cent of total function.

TABLE III. AVERAGE BRONCHOSPIROMETRIC VALUES

		PER CENT OF TOTAL FUNCTION PERFORMED BY THE LUNG OPERATED ON						RATIO OF POSTOPERATIVE TO PREOPERATIVE VALUES LISTED IN COLUMNS TO THE LEFT, PER CENT		
		VITAL CAPACITY		MINUTE VENTILATION		OXYGEN UPTAKE				
NUMBER OF SEGMENTS REMOVED	SUBJECTS	PREOPERATIVE	POSTOPERATIVE	PREOPERATIVE	POSTOPERATIVE	PREOPERATIVE	POSTOPERATIVE	VITAL CAPACITY	MINUTE VENTILATION	OXYGEN UPTAKE
1	7	44	40	43	40	41	37	88	91	90
2*	4	59	48	56	47	51	45	80	81	92
3	5	48	40	51	43	47	35	82	86	78
4-5	4	44	36	43	41	39	32	81	94	84

*High preoperative values attributed to prominent contralateral disease in 2 patients.

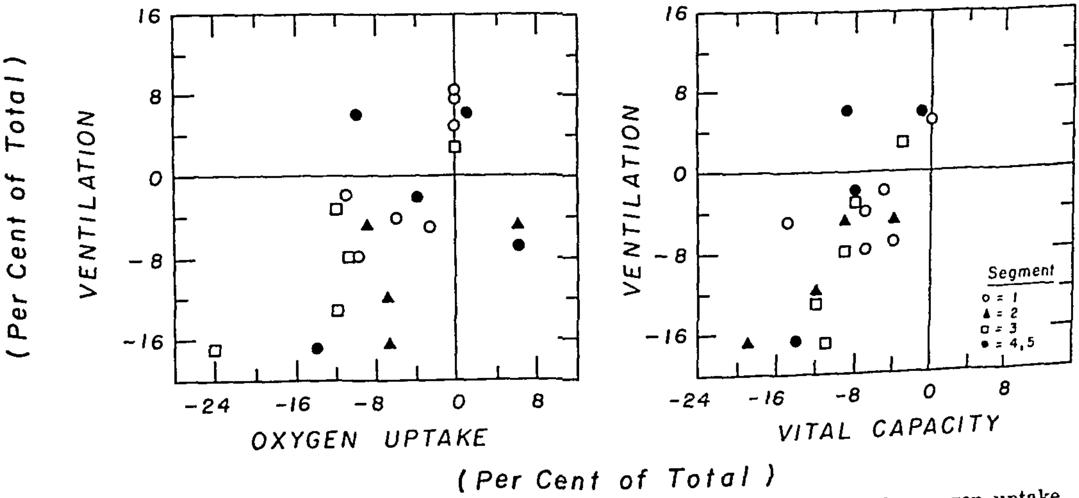

Fig. 4.—Relation of change in ventilation of the lung operated on to changes in oxygen uptake and vital capacity.

should lead to reduction in function, depending on the number removed. By further contrast, our older tuberculous patients had as good functional result as the younger, whereas greater impairment has been reported after resections for bronchiectasis in older patients.[7]

There has been much concern over whether the increased expansion of the remaining pulmonary tissue that occurs after large resections represents or may lead to emphysematous changes. The reduction of functional residual capacity by about 20 per cent of the preoperative values after resection of four or five of the eighteen bronchopulmonary segments indicates the absence of an average increase of expansion of all remaining segments. However, this does not preclude a relatively greater expansion of the segments remaining on the side operated on than on the other. This condition would seem to be better designated as "compensatory overexpansion" than as "compensatory emphysema." Although after operation a minority of our patients had small increases of the residual volume/total capacity ratio, we do not feel justified in considering this as evidence of obstructive emphysema in the absence of obvious abnormalities of the distribution of alveolar ventilation, as indicated by the nitrogen washout index, or of evidence of expiratory obstruction, or of significant increases of absolute residual volume.

Despite the recognized limitations of the tests used to assess pulmonary function, it seems probable that, when the remaining lung is not deficient, the reductions of up to 20 per cent of various lung volumes that follow resection of one to five segments for pulmonary tuberculosis cannot be presently considered to represent a clinically significant impairment.

SUMMARY

To gain further knowledge of the effects of pulmonary resection, pulmonary function studies were performed on 23 adult tuberculous patients about 2 weeks before and 2 to 6 months after resection. There were 12 men and 11 women, with a mean age of 39 years. One to five bronchopulmonary segments were removed without additional space-filling procedures, following a period of combined chemotherapy and modified rest in bed averaging 8 months.

Postoperative changes varied within each of the groups classified according to the number of segments removed. The vital capacity was reduced in almost all cases, the average reduction being similar in all groups. In contrast, the average residual volume was unchanged in patients having one or two segments removed but was reduced in those having three to five segments removed. The total capacity was reduced in all groups but not in close relation to the number of segments removed. In all groups the average maximal breathing capacity and the per cent of rapid vital capacity expired in one second were unchanged postoperatively. The 7-minute alveolar nitrogen (breathing oxygen) was within or near normal limits in most cases before and after operation. The arterial oxygen saturation was normal in each case at rest and with mild exercises preoperatively and postoperatively.

Bronchospirometry, performed in 20 patients, showed [illegible] o [illegible] oss of function could be explained in most instances by unila

The reduction of oxygen uptake by the lung operated on is usually greater than the reduction of its minute volume. There is no close relationship between the reduction of function of the lung operated on (see Fig. 2) and the number of segments (up to four or five) removed.[3, 21] Although the average reduction of oxygen uptake by the lung operated on was found to be greater after lobectomy than after segmental resection by van der Drift,[4] this was usual only with the left upper lobe.

The arterial oxygen saturation was found to be usually normal after lobectomy or lesser resections for bronchiectasis and other lesions[7, 20] and was normal in all of our patients at rest or during exercise or both.

This and similar studies have attempted to determine the extent by which surgical procedures further alter the function of a diseased lung. The preoperative impairment shown by tests of both lungs and of the lung to be operated on varied markedly between patients and was not closely related to the number of segments ultimately removed. This was not surprising. The resections were performed to prevent recurrence of active tuberculosis by removing the danger of reactivation of a residual area of disease following chemotherapy. Very likely, previous involvement of portions of the lungs other than the circumscribed area removed had left its mark in functional impairment. This more diffuse involvement with healing and varying degrees of fibrosis would not necessarily be related to the number of segments containing caseous residues. Only that amount of pulmonary tissue necessary to remove significant residue was resected, as much grossly normal tissue as possible being preserved. Thus the surgeon would hope ideally to remove little functioning pulmonary tissue. If such were possible, and barring major complications, there might be little functional impairment following operation, and the functional status, relative to the preoperative conditions, should be rather uniform despite a varied number of segments removed. There might also be a rather uniform extent of alteration caused by changes in the thoracic wall and pleura. Such changes have been given the term of ''thoracotomy effect.''[3, 7]

It seems clear that the magnitude of reduction of pulmonary volumes is not greatly different after resection of one or several segments for tuberculosis. This suggests that a minimal, rather uniform, amount of functioning lung is in fact removed. In addition, some possible variability related to the number of segments removed is probably masked by the ''thoracotomy'' effect described as following enucleation of small nodules. However, there is a trend toward increasing reduction of pulmonary volumes that indicates that some sacrifice of ventilated lung is made. This trend is likely to be followed when a patient undergoes an additional resection.

Reduction in ventilatory capacity after resection for bronchiectasis was found by Smith and associates[7] to be proportional to the number of resected segments. That such a relationship is less evident after resection for tuberculosis than after resection for bronchiectasis may perhaps be explained by the fact that resection for bronchiectasis is directed at diseased bronchi. Parenchyma distal to some of the diseased bronchi might be contributing significant amounts to total function of the lung. Resection of these bronchopulmonary segments

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Changes were similar in subjects more than or less than 40 years of age and in both sexes.

The reduction of pulmonary function is not greatly different after resection of one or several tuberculous pulmonary segments, suggesting that a minimal, rather uniform, amount of functioning lung is removed with the diseased tissue. Some possible variability related to the number of segments removed is probably masked by the "thoracotomy effect" described as following enucleation of small pulmonary nodules. However, there is a trend toward increasing reduction of pulmonary volumes, indicating that some sacrifice of ventilated lung is made. This trend is likely to be followed when a patient undergoes an additional resection.

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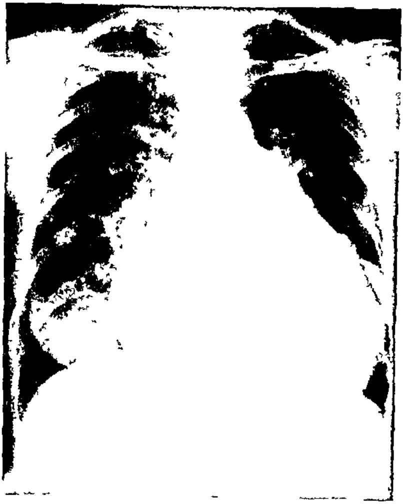

Fig 1 —Roentgenogram of thorax

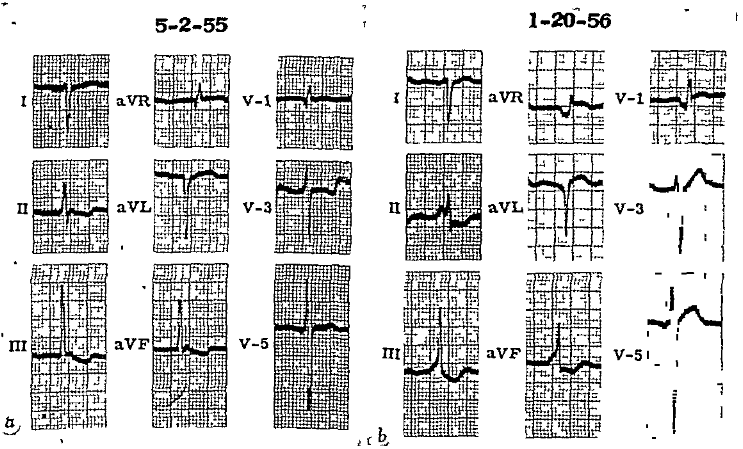

Fig 2 —a, Electrocardiogram made on May 2, 1955 b, Electrocardiogram made on Jan 20, 1956

CONGENITAL COMMUNICATION BETWEEN THE RIGHT CORONARY ARTERY AND THE RIGHT ATRIUM

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LESIONS which cause continuous precordial murmurs that are to be distinguished from patent ductus arteriosus have had considerable coverage in the literature.[1,2] Particularly is this true of aorticopulmonary septal defect,[3-12] ruptured aneurysm of an aortic sinus,[13,14] and ventricular septal defect associated with aortic insufficiency.[15-19] Less emphasis has been made of yet other conditions such as arteriovenous communications, involving vessels of the thoracic cage,[20,21] the subclavian[22,23] or the internal mammary vessels,[24] and communications between the coronary arterial system on one hand and the coronary veins, a cardiac chamber, or the pulmonary trunk, on the other.

Herein is reported the case of an adult patient who had a communication between the right coronary artery and the right atrium. The cardiac failure exhibited is considered to have been caused by the arteriovenous shunt which accompanied the abnormal congenital vascular connection.

It is pertinent to emphasize, by the report of this case, that the coronary arterial system may be involved in congenital conditions which are, or which function, essentially like arteriovenous fistulas. Recognition of the condition in a given patient may lead the way to surgical obliteration of the abnormal communication. Such goals have been accomplished in isolated instances.[25-27]

CASE REPORT

Pertinent Clinical Features.—A woman, first noted to have congestive heart failure at the age of 48 years, was given appropriate treatment for this condition during the ensuing 5 years. At the end of this time, however, she died of intractable failure of the heart. Her history was noncontributory. She had not known that she had heart disease until she consulted a physician for complaints related to the heart failure.

The pertinent facts of her physical examination follow: The heart was greatly enlarged to the left. The point of maximal impulse was in the left anterior axillary line at the fifth and sixth intercostal spaces.

A loud, harsh, blowing, continuous precordial murmur was present. It was heard best anteriorly in the right fifth intercostal space. It also was loud over the lower sternal region. There was enlargement of the cervical veins and of the liver. On occasion, pulsation was noted in the latter organ.

On all examinations the patient was normotensive. A representative blood pressure reading was 110 mm. Hg systolic and 70 mm. diastolic. Usually, the rhythm was regular. Occasionally, ventricular extrasystoles were noted.

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The heart was enlarged, weighing 500 grams. The enlargement was contributed mainly by hypertrophy of each ventricle. In its anterior wall, the right ventricular myocardium measured 0.5 c.c. in thickness; in its posterior wall it measured 0.8 c.c. The left ventricular wall measured 1.7 c.c. in thickness.

The sites of origin of the coronary arteries from the aorta were normal.

The right coronary arterial ostium was wide, measuring 6 mm. Immediately on exit from the aortic wall, the right coronary artery widened to a diameter of about 1.5 c.c.

The proximal part of this artery was tortuous to a point about 3 c.c. from the aortic wall. Here the right coronary artery divided into two branches (Figs. 3 and 4). One branch, 4 mm. in diameter, was essentially a continuation of the right coronary artery in the right atrioventricular sulcus. The other branch was directed posteriorly toward the anterior wall of the base of the right atrial auricular appendage. It was tortuous and had essentially the same appearance as the proximal part of the parent right coronary artery.

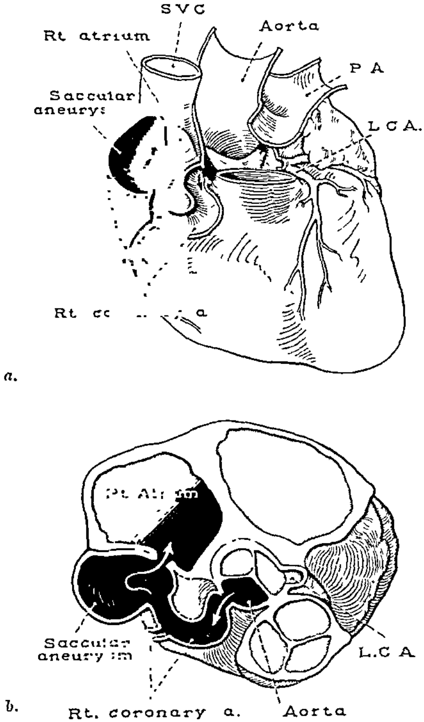

Fig 4.—*a*, In the case reported, the dilated coronary artery originated from the aorta and then divided into a wide branch which in turn became aneurysmally dilated and communicated with the right atrium. The other branch continued as the right coronary artery.

b, The perspective essentially equivalent to that shown in Fig. 3, *a*, wherein a branch of the right coronary artery communicates with the right atrial cavity.

As this branch approached the anterior wall of the right atrium, it dilated suddenly into a saccular aneurysm measuring about 3.5 by 5 cm. Whereas the tortuous porti

A roentgenogram of the thorax (Fig. 1) revealed evidence of cardiac enlargement, rounding of the right margin, and prominence of the pulmonary trunk. The electrocardiogram was difficult to interpret because of the probability of an anomalous excitation of the ventricles of the Wolff-Parkinson-White type (Fig. 2, *a* and *b*). Evident abnormalities were a right axis deviation, an unusual qR pattern in Lead V_1, and a nondiagnostic biphasic QRS (RS) in the left precordial leads. The degree of change in the ST segment in Leads aV_L and aV_F gave strong indication of ischemic injury of the *anterolateral wall of the left* ventricle.

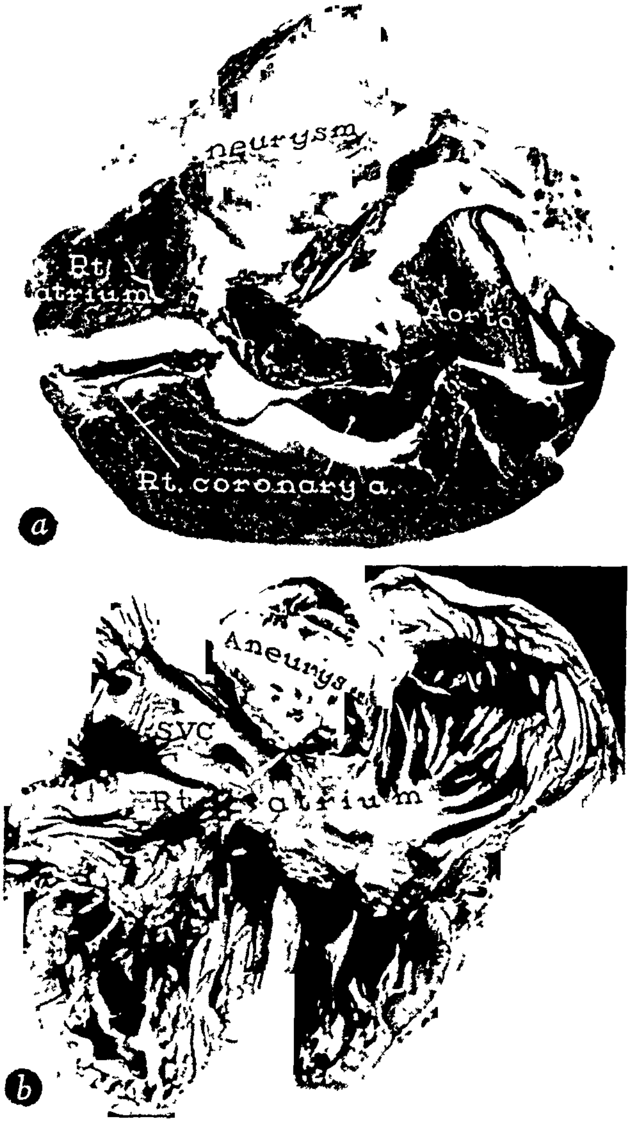

Fig. 3.—*a*, The base of heart viewed from above. A greatly dilated right coronary artery originates from the aorta. In the right atrioventricular sulcus the artery divides. A dilated branch proceeds posteriorly toward the right atrium and has an aneurysm on it in relation to the *right atrium*. The other branch continues in the right atrioventricular sulcus as the right coronary artery.

b, Interior of right side of heart. Shown is one of the communications which connects the right atrium with the aneurysmal part of the dilated branch of the right coronary artery (probe). The junction of superior vena cava and right atrium is indicated as *SVC*.

Pathologic Features.—At necropsy, significant findings were found in relation to the heart which showed anomalous communication of the right coronary artery with the right atrial cavity.

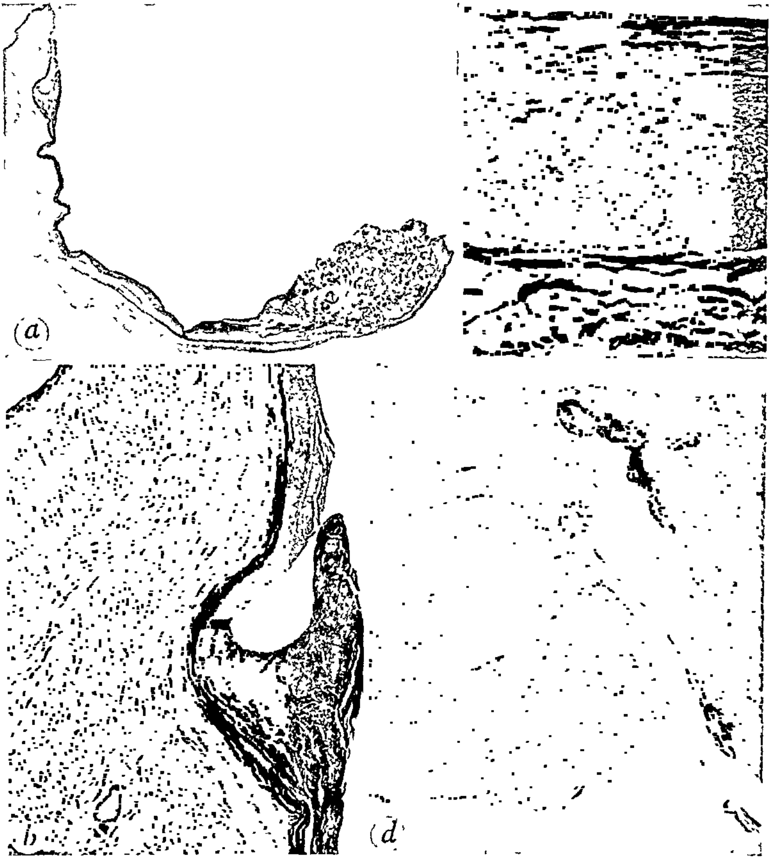

Fig 5.—*a*, The aneurysm of the dilated branch of the right coronary artery. At one zone there is normal arterial structure which suddenly interrupts and beyond this there is nonspecific wall of the saccular aneurysm (see *b*, for details). Calcific material, which may be thrombotic in origin, is deposited on the inner surface of the aneurysm (Verhoeff's elastic tissue stain counterstained with van Gieson's connective tissue stain; ×2).

b, Higher magnification (×30) of junction of arterial wall and aneurysm illustrated in *a*

c, Section through the dilated portion of the proximal segment of the right coronary artery shows evidence of medial hypertrophy. The internal and external elastic laminae are interrupted and represented only by fragments of elastic tissue. There is mild nonspecific fibrous thickening of the intima (Verhoeft's elastic tissue stain, van Gieson's connective tissue stain; ×100).

d, Lateral wall of the left ventricle. Within the myocardium are dilated spaces, some of which are thick-walled sinusoids; others are arteries. A relatively wide sinusoid communicates with the left ventricle shown in the lowermost portion of the illustration (Verhoeff's elastic tissue stain and van Gieson's connective tissue stain; ×5).

right coronary arterial system were thin, the saccular aneurysm had a tough gray fibrous wall containing calcium. Through its posterior wall, the saccular aneurysm communicated with the base of the right auricular appendage by way of three small openings, the largest of which was 3 mm. in diameter.

After giving off the dilated right atrial branch as described, the right coronary artery continued in a normal course and terminated as the posterior descending coronary artery. Just before entering the posterior interventricular sulcus the right coronary artery gave off a branch to the posterior lateral aspect of the left ventricle.

As is often found normally, an accessory artery distributed to the anterior wall of the outflow tract arose from the aorta just anterior to the origin of the main right coronary artery.

The left coronary artery measured 3 mm. at its origin, was devoid of disease, and terminated normally into the anterior descending and left circumflex branches. The terminal branches of the left circumflex coronary artery were distributed to the lateral wall of the left ventricle. Gross connections were not identified between the right and left coronary arteries on gross examination.

Histologic examination of a section taken through the dome of the aneurysm involving the branch of the right coronary artery revealed a small segment of arterial structure which was interrupted and then became continuous with the wall of the aneurysm (Fig. 5, *a* and *b*). The aneurysmal wall was composed of collagenous connective tissue and did not have identifiable arterial elements. The lining of the aneurysm was greatly thickened by the presence of many calcified amorphous masses. These masses had the appearance of calcified thrombotic material. No foam cells or cholesterol clefts were present.

A section taken through the dilated portion of the right coronary artery showed the media to be greatly thickened with muscle (Fig. 5, *c*). The elastic laminae were considerably disrupted and were represented only by occasional strands. The lining of the vessel in this portion was somewhat thickened by nonspecific fibrous tissue. The right coronary artery beyond its dilated portion and the anterior descending coronary artery did not show any remarkable features histologically.

The myocardium in the lateral portion of the left ventricle contained many wide spaces filled with blood (Fig. 5, *d*). Some had walls composed of connective tissue lined by layers of collagen and elastic tissue. These vessels appeared to be dilated sinusoids and some communicated with the left ventricular cavity. Other wide vessels in this region were obviously arteries.

COMMENT

In this case, the communication of a branch of the right coronary artery with the right atrium is representative of a condition described in a number of cases in the literature in which a coronary artery communicates with the right atrium, the right ventricle, or a coronary vein.[28-38]

At operation, Davis, Dillon, Fell, and Gasul[26] observed a branch of the left coronary artery communicating with the right ventricular cavity. Their 19-year-old patient had had a murmur that was suggestive of patent ductus arteriosus. Cardiac catheterization revealed arterialization of blood in the right ventricle. The anomalous communication was interrupted successfully.

In yet another case which was observed by Gasul, Fell, and Dillon,[39] a 9-month-old child had a continuous systolic and diastolic murmur heard best over the fourth and fifth intercostal spaces between the apex and the left sternal border. Because of the unusual location of the murmur it was considered that the child might have an arteriovenous communication involving the coronary system. An angiocardiogram revealed marked enlargement of the

The exact anatomic diagnosis of an arteriovenous communication, wherein a coronary artery takes part in the abnormal flow of blood, is not necessarily simple to make clinically The classic findings at cardiac catheterization would be expected to give rise to evidence of an increased amount of oxygen in the blood in the chamber receiving the abnormal shunt In the case of Davison and associates,[39] wherein the branch of the left circumflex coronary artery communicated with the coronary sinus, cardiac catheterization revealed arterialization of the blood in the right atrium Such a finding coupled with abnormal murmurs of the type usually found in this condition also might be associated with a ruptured aneurysm of an aortic sinus Retrograde aortography probably is a more exact method than cardiac catheterization for diagnosing the exact type of communication [40]

Cases with the foregoing types of communications have basic functional features in common with connections between a main stem coronary artery and the pulmonary trunk [44-49] Moreover, in the condition in which one coronary artery arises from the pulmonary trunk while the other arises from the aorta, present day evidence[50-52] supports the old concept of Brooks[49] that an arrangement functionally similar to that in arteriovenous communications also exists

Abnormal communication of a coronary artery with a right sided cardiac chamber is expected to yield evidence of arterialization in the right side of the heart less commonly a coronary artery communicates with a left sided cavity This results in findings that might be confused with aortic insufficiency In the case of Lovitt and Lutz,[53] a branch of the anterior descending coronary artery communicated with the left ventricular cavity

In Mozen's case,[37] an abnormal communication between the left coronary arterial system and the left atrium was recognized at operation Interruption of the anomalous channel led to disappearance of associated murmurs and thrill

An addendum to a paper by Paul and associates[43] presents a surgical experience of Dr Robert E Gross with a 16 year old boy in whom a clinical diagnosis of patent ductus arteriosus and healed bacterial endarteritis had been made At operation, a portion of the lateral wall of the left ventricle was found to be thin, bulging, and associated with a thrill Though the situation of the major coronary arteries leading to this area was not ascertained, a postoperative diagnosis of "arteriovenous aneurysm, probably of the coronary system" was made

Reid[54] described a gross communication between the apical portion of the left ventricle and the anterior descending coronary artery in the heart of an ox A single opening is often described at the site of an anomalous connection in human beings, but at times there are several, indeed multiple, openings Wilson and Grant[55] found a communication between the right coronary artery and the single ventricle of a malformed heart In this case there was an aneurysm of the artery Histologic examination revealed that the aneurysm opened into many blood filled spaces within the myocardium These spaces, in turn opened into the ventricular cavity right coronary artery which appeared to end in a large dilated opacified region close to the cardiac apex. Cardiac catheterization revealed normal intracardiac pressures and evidence of a small left-to-right shunt at right ventricular level. At operation, a greatly dilated right coronary artery was found to enter the diaphragmatic portion of the right ventricle near the apex. The vessel was interrupted and the patient's course subsequently was uneventful.

In an adult patient with a continuous precordial murmur, Morrow[40] observed, on retrograde aortography, a communication between the aorta and the right atrium. At operation an anomalous communication between the right coronary artery and the right atrium was identified. The communication was closed at its right atrial aspect.

In the study of a human embryo, Essenberg[41] found that a branch of the left coronary artery penetrated the wall of the left atrium and then coursed through the cavity of the left atrium for a short distance and penetrated the right atrial wall. This vessel may have communicated with the right atrial cavity but the serial sections to determine this point were incomplete.

In the case of an adult patient, Garomella[42] observed that, at necropsy, a connection existed between an accessory branch of the left coronary artery and the right atrium.

Paul and associates[43] described the case of a 9-year-old boy with a continuous precordial murmur who, at operation, was found to have an enlarged right coronary artery that seemed to communicate with a tributary of the coronary sinus.

A common finding in the group of cases mentioned was the presence of a left-to-right shunt from a coronary artery into some point in the lesser circulation. Such a communication may give rise to one or more of the several complications of arteriovenous fistula anywhere in the systemic circulation. If the opening between the coronary artery and the right-sided chamber is sufficiently wide to cause a large shunt, the latter may be responsible for cardiac failure. The dynamics of arteriovenous fistula are such that bacterial endarteritis may occur in the abnormal channel.[36, 38]

An additional complication of the arteriovenous communication is that of ischemia beyond the point of the abnormal communication. The resulting myocardial ischemia may give rise to a picture which may simulate coronary insufficiency resulting from the common type of coronary disease, namely, atherosclerosis.

The abnormal vibrations generated by the arteriovenous shunt have frequently been described as the cause of a continuous thrill or of a to-and-fro murmur associated with a continuous thrill. Although in most instances wherein such a murmur was described, the location of the murmur was farther to the right and lower than that of typical patent ductus arteriosus, the latter condition has been suspected in some patients, as in the case of Davis and associates.[26] In some cases, such as the case of Espino Vela and associates,[38] the position of the murmur has suggested another type of arteriovenous communication, namely, an aneurysm of an aortic sinus (Valsalva) with rupture into a right-sided cardiac chamber.

that exist between the coronary arteries and the cardiac chambers. In the case we have reported, the communication between the right coronary artery and the right atrium may therefore be considered a normal connection. The abnormality resides, therefore, not in the fact that connections of the coronary artery with the atrial cavity exist, but rather in the large size of the connections. Normally the communications should have been reduced to microscopic size. A similar view was expressed by Grant[57] for the abnormal communication between a coronary artery and a cardiac ventricle in the case previously reported by Wilson and Grant.[55]

SUMMARY

The case is reported of a woman having anomalous communication between the right coronary artery and the right atrium. Congestive cardiac failure was considered to be a complication of a left-to-right shunt. Dilatation of the anomalous arterial channel was complicated by the formation of a localized saccular aneurysm.

In creating a basis for an arteriovenous-like condition, the malformation in this case is representative of other cases in which the coronary arterial system communicates with the coronary venous system, a left or right cardiac chamber, or the pulmonary trunk. The continuous murmur often present in such conditions may be confused with patent ductus arteriosus or with other conditions which enter into the differential diagnosis of this malformation.

Surgical obliteration 'of arteriovenous-like fistulas in which the coronary arteries participate has been accomplished in a few reported cases.

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The association of a saccular aneurysm with an abnormal communication between the coronary artery and the cardiac chamber is frequently described and, moreover, in many reports the aneurysm is given a major position in the title and discussion. The aneurysm, however, is to be viewed simply as a complication of an arteriovenous communication. In all cases wherein such communications have been described, the artery proximal to the communication is greatly dilated as it was in the case reported herein. Associated with the right coronary arterial dilatation in our case was hypertrophy of the media of the artery proximal to the anomalous communication, a feature which is frequent in arteries proximal to arteriovenous communications anywhere in the body. Of interest with regard to genesis of the saccular aneurysm is the fact that associated with the dilatation there was interruption of the elastic layers of the dilated segment of artery. Such interruptions in the elastic layers probably represented the underlying cause for the development of the saccular aneurysm. In the case reported herein, a section of the saccular aneurysm revealed that for the most part it was composed of nonspecific connective tissue. At one point a segment of arterial wall ended abruptly and then communicated with the aneurysmal wall. It is probable, as suggested by Scott,[46] that the saccular aneurysm arose at the site of an arterial branch. The calcific material within the saccular aneurysm in the case reported is possibly derived from thrombotic material within the saccular aneurysm.

In one case the electrocardiogram gave suggestive evidence of ischemia in the anterolateral region of the left ventricle. This is of special interest in view of the fact that, histologically, the lateral wall of the left ventricle showed dilated arteries and sinusoids. This brings up for consideration the possibility that blood in the left ventricular cavity was "running off" through sinusoids into the arterial system and hence into the right atrium through the anomalous communication. Such findings suggest a certain possibility regarding the circulation in conditions wherein a coronary artery participates in an arteriovenous-like communication. The blood escaping abnormally through the anomalous coronary artery may be derived not only from that artery but also from other arteries with which it connects and even from the ventricular chambers.

Mention should be made of the developmental basis for the abnormal communication between the right coronary artery and the right atrium in the case reported herein. From the developmental analysis of the coronary vascular system made by Grant,[56] it is apparent that in the early stages of cardiac development the myocardium is nourished by blood which is brought to it through wide intertrabecular spaces. These exist in the myocardium and are continuous with the cardiac chambers. Later, the coronary veins and the coronary arteries develop and connect with the epicardial aspects of the intertrabecular spaces. Following this phase, the growth of the myocardium causes the intertrabecular spaces to be reduced in size to the caliber of capillaries. The intertrabecular spaces, however, maintain communication with the cardiac chambers and therefore represent, in the adult, the normal small communications

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of the esophageal atresia. Accordingly, on Jan. 3, 1956, an extrapleural thoracotomy was performed. Exploration revealed a complete esophageal atresia of the usual type, with a blind upper segment just below the thoracic inlet and attachment of the distal segment to the posterior aspect of the trachea. The proximal aspect of the distal segment was narrow, firm, and cordlike at the point of tracheal attachment and the absence of the characteristic ballooning on inspiration of the distal segment and the absence of air in the gastrointestinal tract was felt to be on that basis. The attachment to the trachea was divided and a small portion of the divided end sacrificed to secure an adequate lumen for anastomosis. A small probe was passed well into the distal limb and was thought to pass through the esophageal hiatus. Continuity of the esophagus was readily re-established without tension by an anastomosis employing two layers of fine silk sutures. A small, soft drain was placed retropleurally before closure of the chest wall. The child tolerated the procedure well and the immediate postoperative course remained good for approximately 48 hours at which time respiratory distress and cyanosis reappeared and the child suddenly died.

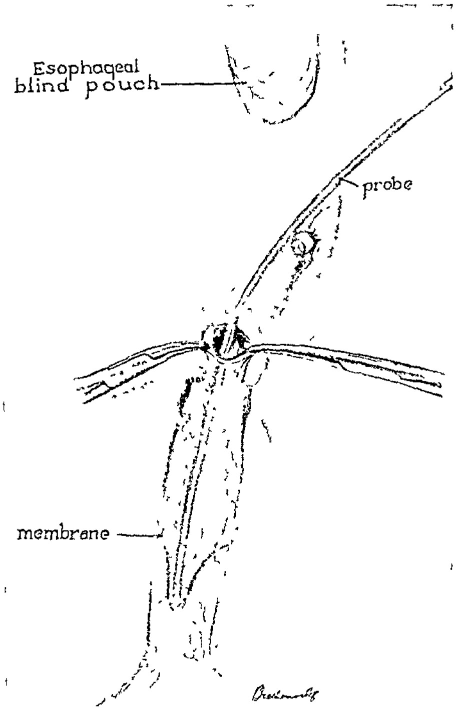

Fig 1.—Artist's concept of the esophageal atresia. The tracheosophageal band has been divided and a proble in the distal segment demonstrates the pliable membranous obstruction.

UNUSUAL ESOPHAGEAL ATRESIA WITH DISTANT MEMBRANOUS OBSTRUCTION OF THE ESOPHAGUS

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INCREASING experience with the surgical correction of esophageal atresia indicates that the majority of infants so afflicted may be saved if other severe anomalies are absent. Fortunately, most babies with esophageal atresia and associated tracheoesophageal fistula have no other important abnormalities. If such serious anomalies as congenital heart disease and malformation of the anus and rectum are present, they are readily recognized and do not often affect the initial treatment of the child.[5] Although rare, other anomalies do occur which affect the initial surgical management and the possibility of their presence should be constantly borne in mind. We have recently encountered such a combination and the unusual characteristics of the second abnormality prompted this report.

CASE REPORT

E. M., a 13-hour-old white female, was admitted to Jefferson Davis Hospital, Houston, Texas, on Dec. 31, 1955, because of difficulty in respiration, retraction of sternum, and transient cyanosis. These respiratory difficulties had ensued at birth in another hospital. Physical examination revealed a fairly well-developed, premature child, weighing 1,600 Gm., in mild respiratory distress. Increased respiratory effort with retraction of lower sternum was present, but cyanosis occurred only with crying. Expansion and breath sounds were equal bilaterally. Examination of the heart revealed marked cardiomegaly, a forceful precordial impulse with systolic thrill and a loud, harsh systolic murmur heard best in the left parasternal area and transmitted to the anterior and left posterior aspects of the chest. The liver was palpable below the costal margin. Initial laboratory studies were within normal limits. Roentgenograms of the chest revealed a markedly enlarged, globular-shaped heart. Electrocardiographic studies demonstrated right ventricular hypertrophy with severe ischemic changes. A diagnosis of congenital heart disease with an interatrial septal defect and/or interventricular septal defect was made. When further cyanosis and respiratory distress occurred, congestive failure was suspected and the child was digitalized. Some improvement followed but it soon became apparent that, in addition to respiratory distress, the child had excessive drooling of saliva and when oral feedings were begun regurgitation of all feedings occurred. Diagnosis of esophageal atresia was confirmed by the inability to pass a catheter through the esophagus and roentgenographic evidence of a blind upper esophageal pouch following instillation of 1 c.c. of Lipidol. No air was demonstrable in the gastrointestinal tract and it was felt that the atresia was not associated with tracheoesophageal fistula. Although some respiratory difficulty persisted, the general condition seemed improved enough by the third day of life to warrant surgical correction

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everting the distal segment or by a small esophagotomy at a lower level with resection of the web. The demonstration of aspirated bile through a catheter into the stomach would have circumvented the error and we have since added this maneuver to the management of these cases. Aspiration of the distended stomach may also be carried out at this time. Since hypertrophic pyloric stenosis and duodenal obstruction are frequently associated anomalies,[2, 6, 9, 10, 11] demonstration of bile in the catheter is evidence of patency of the pylorus and upper duodenum.

SUMMARY

An unusual case of esophageal atresia with bandlike attachment to the trachea associated with complete membranous obstruction in the distal esophageal segment is presented. At operation for tracheoesophageal fistula a catheter should be introduced into the distal esophageal segment and into the stomach to determine the presence of such a membranous obstruction, which can be easily corrected if found. The aspiration of bile will also exclude hypertrophic pyloric stenosis and duodenal obstruction which are occasional associated anomalies.

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Post-mortem examination confirmed the suspected presence of severe congenital heart disease manifest by great dilatation and hypertrophy of the right atrium and ventricle, large atrial and ventricular septal defects, severe pulmonary stenosis, and a patent ductus arteriosus. The right pleural cavity was dry, the lung well expanded and the esophageal anastomosis intact. Upon removal of the esophagus and further inspection, the presence of a completely unsuspected congenital esophageal web was found in the lower esophageal segment approximately 2 cm. above the esophageal-gastric junction. Probing of the lumen of the distal segment, as was done at the time of surgical reconstruction of the proximal atresia, demonstrated that the pliable web could readily be stretched ahead of the probe as far as the stomach, thus giving an erroneous impression of patency of the distal segment (Fig. 1). The remainder of the necropsy findings were not remarkable with the exception of congestive changes in the lungs and a liver compatible with congestive heart failure.

DISCUSSION

Esophageal atresia may assume a variety of forms either with or without associated tracheoesophageal fistula; however, the majority occur in a characteristic pattern consisting of a blind upper segment and a lower pouch connecting with the trachea. Because of the frequency of this type of anomaly, treatment has become relatively standardized with significant improvement in results.

Despite the large number of cases of tracheoesophageal fistula now reported in the literature, the occurrence of a membranous obstruction of the esophagus in association with complete atresia and tracheoesophageal fistula has not been described. Theoretically, a membranous form of atresia might occur in the esophagus as it does in other portions of the gastrointestinal tract but membranous obstruction per se is quite rare. MacKenzie,[7] Tenon,[12] Rossi,[8] and Abel[1] have described membranous obstruction and it is likely that many of the cases of congenital stenosis represent incomplete webs or membranes. The only recorded instance of membranous obstruction associated with tracheoesophageal fistula was the interesting case of Schwartz and Dale.[9] In this case, continuity of the esophagus was not interrupted and the fistulous connection was distal to the web. Gross[3] encountered a stenotic area in the distal segment of one case in their large series of atresia and associated tracheoesophageal fistula, but no case of membranous obstruction associated with proximal atresia and tracheoesophageal fistula has been reported.

The presence of a weblike membrane in the distal segment of our case was completely undetected by the usual maneuver of passing a probe or catheter into the stomach. The elasticity of the structure allowed it to be readily pushed ahead of the probe, a maneuver we were able to duplicate with the post-mortem specimen (Fig. 1). Fortunately, the outcome of this particular case was not influenced by this additional anomaly but, had the child survived, an additional operative procedure would have been required. The existence of a second obstruction was considered and sought because of the absence of air in the gastrointestinal tract but the demonstration of the cordlike nonpatent attachment to the trachea, a condition previously described,[4] was considered an adequate explanation for the absence of air. The secondary obstruction could have been readily corrected at the time of the original procedure by

TABLE I. INCIDENCE OF SECONDARY MEDIASTINAL NEOPLASMS

PRIMARY NEOPLASM	NUMBER OF CASES OF SECONDARY NEOPLASMS IN THE MEDIASTINUM
Carcinoma of breast	73
Carcinoma of bronchus and lung	60
Carcinoma of esophagus	35
Carcinoma of stomach	35
Carcinoma of pancreas and colon, each	12
Carcinoma of urinary bladder	10
Carcinoma of prostate	9
Carcinoma of gall bladder, kidney, and unknown etiology, each	7
Carcinoma of uterus and ovary, each	6
Carcinoma of liver and thyroid, each	5
Malignant melanoma	5
Sarcoma of extremities, etc.	5
Sarcoma of retroperitoneal region	4
Carcinoma of rectum	3
Carcinoma of extrahepatic bile ducts	2
Carcinoma of skin and tongue, each	2
Osteogenic sarcoma of femur	2
Sarcoma of unknown origin	2
Carcinoma of trachea, pharynx, larynx, buccal mucosa, left ureter, penis, vagina, each	1
Sarcoma and leiomyosarcoma of esophagus, each	1
Reticulum cell sarcoma of stomach	1
Melanosarcoma of liver	1
Mixed tumor of kidney, left	1
Myeloma	1
Fibrosarcoma of unknown etiology	1
Neuroblastoma of spinal nerve roots	1
Adenocarcinoma and sympathogonioma of adrenal gland, each	1
Testicular tumors: seminoma type (?), malignant, teratoma, and carcinoma, each	1
Total	337

TABLE II. INCIDENCE OF SECONDARY MEDIASTINAL NEOPLASMS BY DECADES

	DECADES					
	1895–1910	1911–20	1921–30	1931–40	1941–50	TOTALS
Number of Autopsies	*2,133*	*1,336*	*1,856*	*1,722*	*2,201*	*9,248*
Breast	0	4	11	15	43	73
Bronchus and lung	3	0	10	13	34	60
Esophagus	8	1	14	7	7	37
Stomach	4	1	7	12	12	36
All Others	14	10	20	34	53	131
Totals	29	16	62	81	149	337

are given in Table III. Only the cases of metastases of the more common primary lesions are included. In some cases it was impossible to decide which lymph node group were involved as the protocols referred only to mediastinal lymph nodes and not to their exact location. For the sake of completeness, these cases, worthy of minor consideration, are enumerated at the end.

In addition to learning which of the mediastinal lymph nodes contained metastases, an attempt was made to determine in which cases the involvement could have been recognized clinically, as by gross examination at the time of a thoracotomy. Several criteria were used for making these decisions. One was the consistency and, another, the size of the lymph nodes involved. The

SECONDARY NEOPLASMS OF THE MEDIASTINUM

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DEVELOPMENTS and progress in the field of oncology have frequently resulted from investigations and considerations of the natural history of neoplastic diseases. Since some secondary neoplasms of the mediastinum are a part of the clinical course of several surgically treated malignant tumors, and since they have not been studied extensively, an investigation of them was initiated in 1951.

MATERIAL AND RESULTS

Protocols of autopsies performed in general hospitals over a period of years offered the best source of material for such a study. From August, 1895, to Dec. 31, 1950, 9,248 autopsies were performed by physicians in the Pathology Department of the Presbyterian Hospital. The anatomic diagnoses of each autopsy were examined in order to learn the presence of a neoplasm. In such cases, the gross description and report of microscopic examinations were scrutinized to learn of possible mediastinal involvement. Lesions of the lymph nodes, such as Hodgkin's disease and lymphosarcoma, were not included in the study. In these cases of generalized disease, it is difficult to decide at autopsy which is the primary or secondary lesion.

There were 337 protocols which recorded neoplasms that had metastases involving the mediastinum. In Table I, the primary neoplasms from which these metastases originated are listed. Carcinoma of the breast, bronchus and lung, esophagus, and stomach, the most common malignant diseases of both sexes, are at the top of the list. The secondary neoplasms of the bronchus and lung were placed in one group because their exact origin cannot always be determined. Forty-five types of primary tumors are included.

As a sidelight, a tabulation of the cases of secondary neoplasms, by decades in the period studied, was made. Table II demonstrates an increasing number of the primary neoplasms with such metastases in the breast, bronchus, and lung. There was an average of 50 more autopsies a year in the 1940's than in the 1930's. This would account for only a portion of the increased incidence of cases of metastases in the last decade studied.

The nomenclature of Rouviere[1] was used to describe the location of the metastases as recorded in the protocols. The lymph nodes, as well as other structures in the mediastinum most frequently involved by the secondary neoplasms,

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In Table I, a large number of primary neoplasms are found to metastasize to the mediastinum Previous lists[3, 7, 8, 13] have been more limited in number A number of primary neoplasms of the abdomen and pelvis, as well as of the head and extremities, are present in this series As a result of this investigation, it is apparent that practically any carcinoma or sarcoma in or on the human body is capable of spreading to the mediastinum In this study, as in the series of Abrams and his associates[9] carcinoma of the breast was the first in order of incidence and carcinoma of the lung was second

The increasing incidence of primary carcinoma of the lung in Table II is obvious and confirms the opinions of these interested in this disease in this country and abroad

Table II also authenticates the opinion that neoplastic disease has been increasing Undoubtedly the significant factor is that more people are living to an older age

Probably the most significant data are compiled in Table III The marked incidence of involvement of the tracheobronchial lymph nodes is noteworthy The nodes of the tracheal bifurcation, or inter tracheobronchial lymph nodes, are a crossroad for lymphatic or lymphoid relays The large number of these nodes involved by secondary neoplasms of the stomach is an exception to previous pathologic studies and reports[4, 13]

It is remarkable that a large number of the involved lymph nodes could be distinguished by their gross appearance (Table IV) However, only a few cases had involvement which could be diagnosed on the usual posteroanterior film The size of 5 cm (in diameter), for the lymph nodes or lymph node masses, was used because a lesion of this size would probably be distinguished from the density due to the spine, cardiac shadow and other mediastinal structures The routine of McCort and Robbins[11], and other radiologists, of utilizing a barium swallow and a Bucky film, or planigrams, should increase the number of clinically diagnosed metastatic lesions of the mediastinum

SUMMARY AND CONCLUSIONS

This is the first known study of the incidence of secondary neoplasms of the mediastinum and of the lymph nodes involved by them

Clinicians, pathologists, and radiologists should be more exacting in their terminology in describing the lymph nodes of the mediastinum Designating these nodes as mediastinal is not satisfactory for the future consideration and management of patients with such metastases

Any malignant neoplasm may metastasize to the lymph nodes of the mediastinum The most frequently found metastases are from carcinomas of the breast, lung, esophagus, and stomach

The fact that many of the involved nodes of the mediastinum can be distinguished grossly, as at the time of thoracotomy, has clinical value However, microscopic metastases are frequently present in grossly uninvolved nodes

TABLE III. LYMPH NODES AND STRUCTURES INVOLVED BY SECONDARY NEOPLASMS IN THE MEDIASTINUM

	PRIMARY NEOPLASM			
	STOMACH	ESOPHAGUS	LUNG AND BRONCHUS	BREAST
Number of cases	*36*	*37*	*60*	*73*
Juxta-esophageal LN	6	16	7	13
Thoracic duct	2	3	2	2
Juxta-aortic LN	11	4	12	16
Pulmonary root LN	1	9	17	21
Anterior mediastinal LN	3	1	14	18
Pericardium	0	–	14	19
Right laterotracheal LN	23	14	38	40
Inter-tracheobronchial LN	19	11	32	32
Left laterotracheal LN	21	12	33	41
Mediastinal LN	10	5	24	31

LN—lymph node.

latter is not necessarily a satisfactory criterion because it is well known that lymph nodes are enlarged by infections. However, used in conjunction with the consistency of the node, as by palpation, it is valuable. In Table IV, these data are summarized.

TABLE IV. CLINICAL DATA CONCERNING LYMPH NODES

	NO. OF CASES	GROSSLY INVOLVED	GROSSLY INVOLVED (?)	SEEN ON X-RAY
Stomach	36	31	0	0
Esophagus	37	32	5	1
Breast	73	60	1	8
Bronchus and lung	60	56	0	16

Another attempt was made, during the analysis of the data, to decide which of the secondary neoplasms could be seen on a routine thoracic roentgenogram. Two criteria were utilized for a positive conclusion. One was the size of the involved node, or nodes, and the other was its location. According to McCort and Robbins,[2] the tracheobronchial and right anterior mediastinal chains are more easily seen than are most of the other nodes of the mediastinum on such a roentgenogram. When the involved node, or nodes, was in the above groups and 5 cm. in diameter, or larger, it was concluded that it could be seen. According to this evaluation, approximately 25 per cent of the secondary neoplasms of bronchiogenic carcinoma could be seen. However, in only one case of carcinoma of the stomach and esophagus could an enlarged node be seen. If these criteria are accurate, less than 15 per cent of the mediastinal metastases from carcinoma of the breast could be outlined on a thoracic roentgenogram.

DISCUSSION

Only recently has there been an attempt by clinicians to utilize the anatomist's descriptions of the lymphatic system of the mediastinum.[10 11 12] The results of the present investigation are published to focus more attention not only on the mediastinum but its chains of lymph nodes and their position in the spread of malignant disease.

RADIATION THERAPY FOR RECURRENT CARCINOMA IN THE BRONCHIAL STUMP

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THE surgical approach to bronchogenic carcinoma has left in its wake an increasing number of recurrences in the bronchial stump This report details a course of management, combining surgery and three full courses of radiation therapy, which allowed such a patient to lead a productive life for 4½ years following the definitive diagnosis of his malignancy

CASE REPORT

The patient (G C M), a 57 year old white man, was first seen in April, 1952, with a history of chronic respiratory infection dating back to 1946 He had been a heavy cigarette smoker for 25 years His cough had increased progressively over the previous 6 weeks He had had fever, dyspnea and occasional hemoptysis Râles and decreased breath sounds were detected at the right base Roentgenograms and fluoroscopy of the chest demonstrated an enlargement of the right root and partial atelectasis of the right lower lobe (Fig 1) Bronchoscopy revealed a large mass in the right main bronchus just below the carina Pathologic examination of biopsy material removed at bronchoscopy showed epidermoid carcinoma

On April 25, 1952, a right pneumonectomy was performed during which the surgeon noted tumor tissue at the line of bronchial transection at the carina (Fig 2) The hilar and mediastinal nodes were enlarged, however, the pathologist reported no tumor found in the nodes removed proximal to the transection Postoperatively the patient gained weight, had less dyspnea, and returned to his work as an executive

In May, 1953, weight loss, cough, and hemoptysis recurred Bronchoscopy on Aug 7, 1953, 16 months post pneumonectomy, revealed recurrent tumor in the right main bronchial stump (Fig 3) Tumor partially occluded the left main bronchus as well In an effort to clear the airway, the bronchoscopist removed as much tumor as possible

On Aug 14, 1953, the patient was started on a course of radiation therapy with the carina as the center of rotation and utilizing a 10 × 10 cm port with a focal axis distance of 100 cm One million volt radiation was employed at 3 Ma and HVL of 9 0 mm of copper Over a period of 26 treatments and 38 days, a tumor dose of 5,200 r was delivered The patient tolerated the treatment well, showing progressive improvement and experiencing only minimal reddening of the skin over the sternum (Fig 4)

The patient did very well thereafter, complaining only of slight dyspnea but no cough fever, or hemoptysis until February, 1955, when he again raised blood streaked mucus On Feb 18, 1955, bronchoscopy again revealed recurrent tumor in the bronchial stump on the right, partially occluding the left main bronchus (Fig 5) The bronchoscopist removed as much tumor as feasible and the patient was begun on a second course of radiation

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Unfortunately, the present routine used in many hospitals of taking a posteroanterior chest film to decide if there are intrathoracic metastases is insufficient. This study demonstrates that in relatively few instances, metastases of the mediastinum can be seen with such a method.

One of the avenues for further progress in oncology lies in more detailed studies and in better management of secondary neoplasms of the mediastinum.

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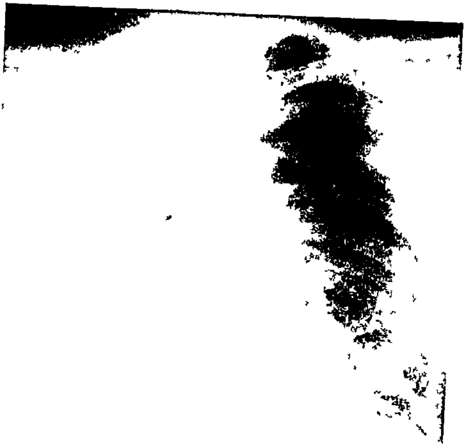

Fig 4—PA roentgen film of chest made on Sept. 15, 1953, 17 months after pneumonectomy and on completion of first course of radiation shows a normal post-pneumectomy chest

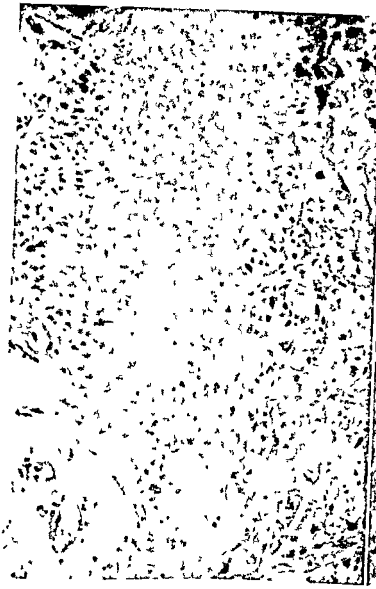

Fig 5

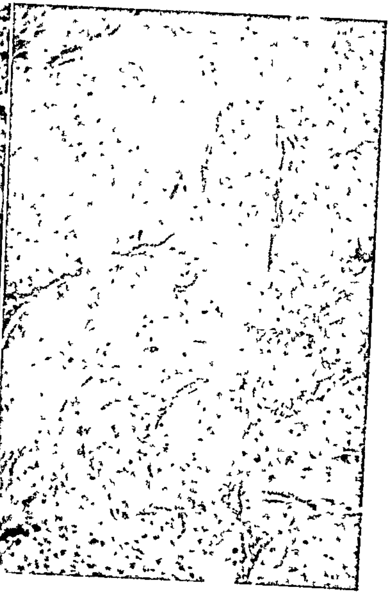

Fig 6

Fig 5—Biopsy of second recurrence on Feb 18, 1955, again shows fairly mature invasive epidermoid carcinoma

Fig 6—Biopsy on June 29, 1956, demonstrates similar recurrent tumor

A.

B

Fig 1.—PA (*A*) and lateral (*B*) chest films made on March 24, 1952, demonstrate atelectatic segment at right base posteriorly. Fluoroscopist noted shift of heart to right on inspiration

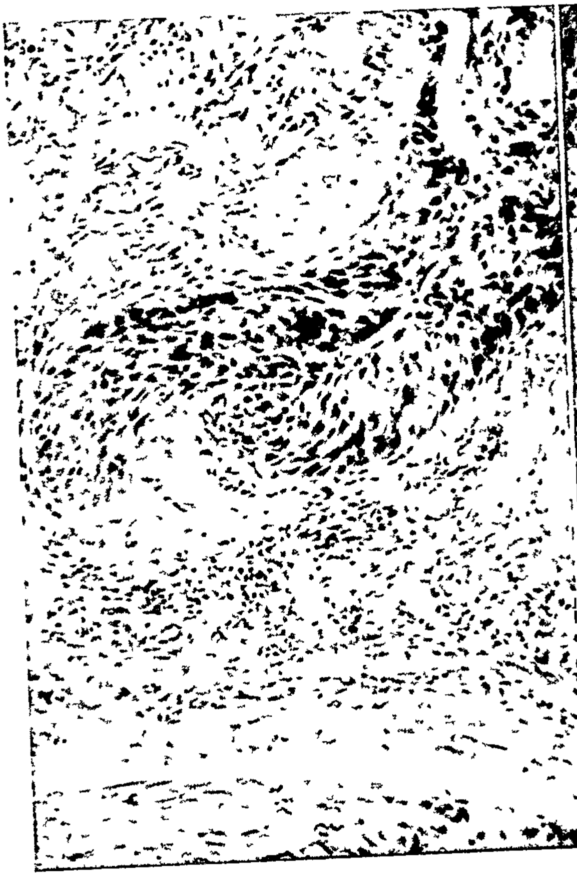

Fig. 2.

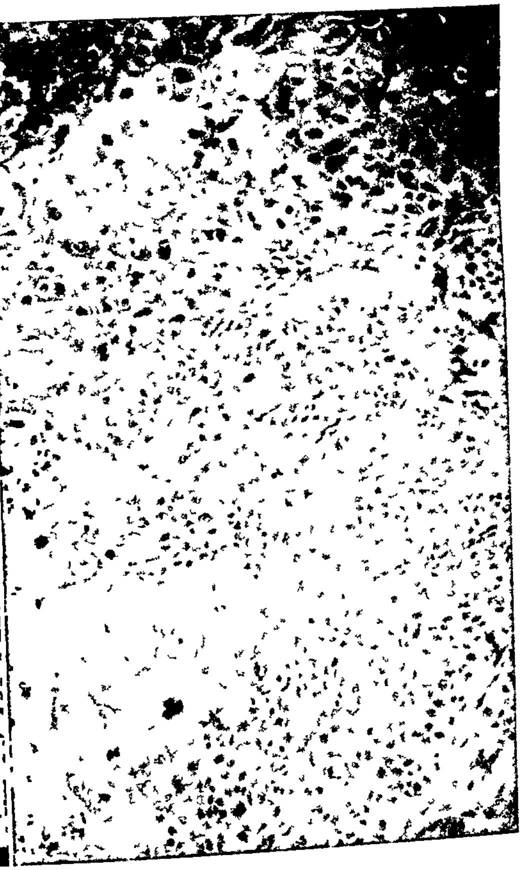

Fig 3

Fig 2.—Section from surgical specimen shows only demonstrable focus of microscopic invasion. Remainder of tumor was papillary in nature

Fig 3.—Biopsy of recurrence on Aug. 7, 1953, shows a fibrous stroma infiltrated with epidermoid cells. The cells appear fairly mature but show some mitoses and variations

therapy on Feb. 23, 1955. The same factors as described above were utilized and over a period of 26 treatments in 36 days, 5,000 r was delivered to the carina. The patient noted diminution of cough and dyspnea and was able to continue at work throughout therapy.

In June, 1956, cough returned along with hemoptysis and dyspnea. Once again the bronchoscopist cleaned out as much tumor as technically possible and obtained pathologic proof of recurrent epidermoid carcinoma (Fig. 6).

The hazards of further radiotherapy were weighed against the patient's otherwise hopeless outlook and on July 10, 1956, a third course of radiation therapy was started, using the factors described above. Over a period of 27 treatments and 37 days, 5,000 r was again delivered to the tumor. The patient continued at his job with some exertional dyspnea and cough, although less than prior to his last course of treatment (Fig. 7).

On Oct 21, 1956, 4 years and 6 months following the original surgery, an upper respiratory tract infection developed and after an initial response to antibiotics and bed rest, he died on Oct. 22, 1956, with a massive pulmonary hemorrhage

At post mortem, an area of necrosis, measuring 4 cm. by 5 cm, was found in the tracheobronchial tree at the location where recurrent tumor had been seen at bronchoscopy prior to therapy. This was thought to represent the source of terminal hemorrhage. No definite evidence of residual tumor cells was found on detailed pathologic investigation of sections through the area of necrosis or elsewhere (Figs. 8 and 9). A moderate degree of radiation fibrosis was present in the remaining lung as well as some pleural fibrosis. Marked tracheitis was also seen

DISCUSSION

Despite vigorous efforts to improve and refine methods of diagnosis and treatment, bronchogenic carcinoma continues to take a toll of approximately 28,000 lives annually and is the second most frequent cause of death due to neoplasm among males in the United States today. In wholly unselected series, the 5 year survival of patients with an established diagnosis of bronchogenic carcinoma is estimated at less than 5 per cent.[1]

Radiation therapy following surgery is seldom prescribed for patients suffering with bronchogenic carcinoma with the expectation of achieving cure, but rather as a palliative measure Usually, the intent of the radiotherapist is to relieve bronchial obstruction and secondary peripheral infection, superior mediastinal obstruction, pain, cough, or acute pulmonary osteoarthropathy.[2] There is considerable debate as to whether radiation therapy actually does increase longevity even when palliation is achieved.[3]

Habein and associates[4] of the Mayo Clinic recently reported on 18 patients with recurrence in the bronchial stump, several of whom received radiation therapy We have had 4 recent cases of such recurrence, one of which is reported. The unusual palliation obtained in this case, employing surgery and repeated courses of radiation therapy, has encouraged us to attempt a similar plan in other cases It is too early to evaluate our results in the 3 cases not reported here

The ease with which this patient tolerated his re-treatment with radiation has raised the question as to whether re-treatment might not also be of value in situations other than bronchial stump recurrence. A dilemma occasionally arises in the patient who has had 18 to 24 months' arrest of bronchogenic carcinoma following radiation therapy and who has tolerated his original course

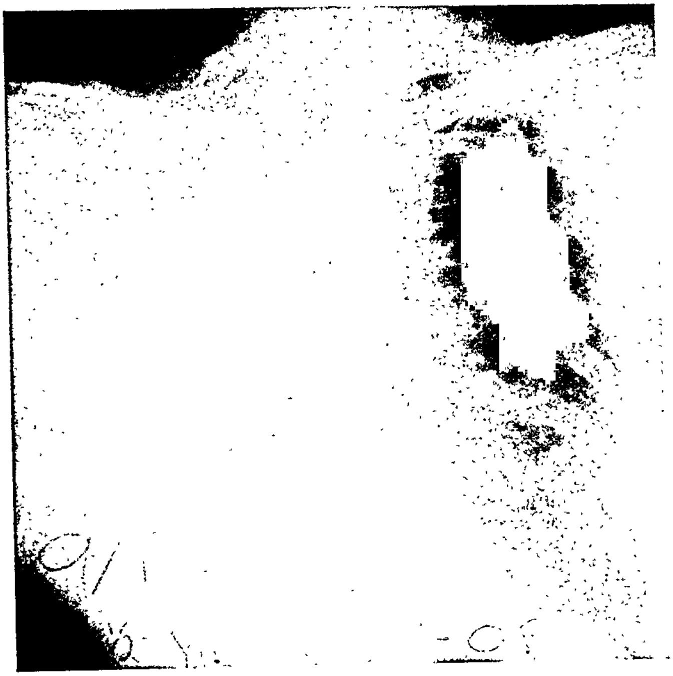

Fig. 7.—PA roentgenogram of chest made on Sept. 12, 1956, 4½ years after operation and after administration of 15,200 r to the carina, shows only normal post-pneumonectomy changes.

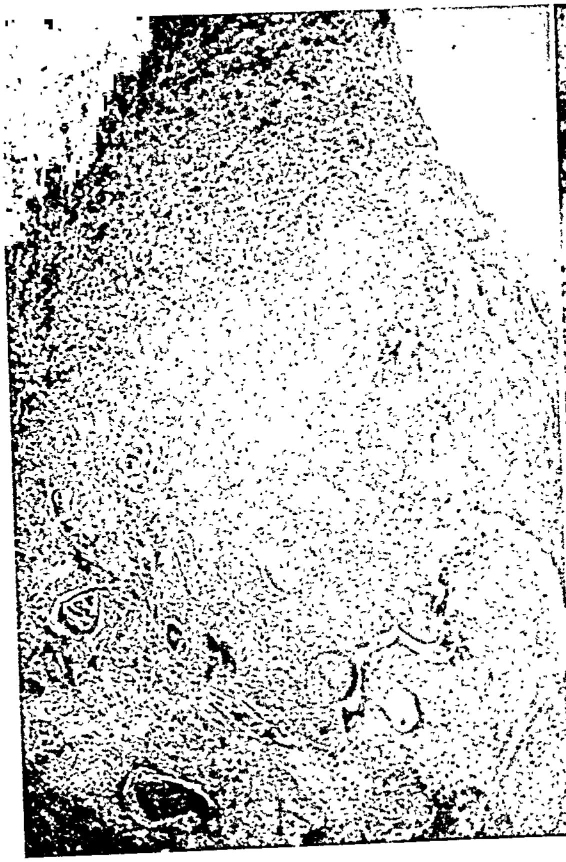

Fig. 8.

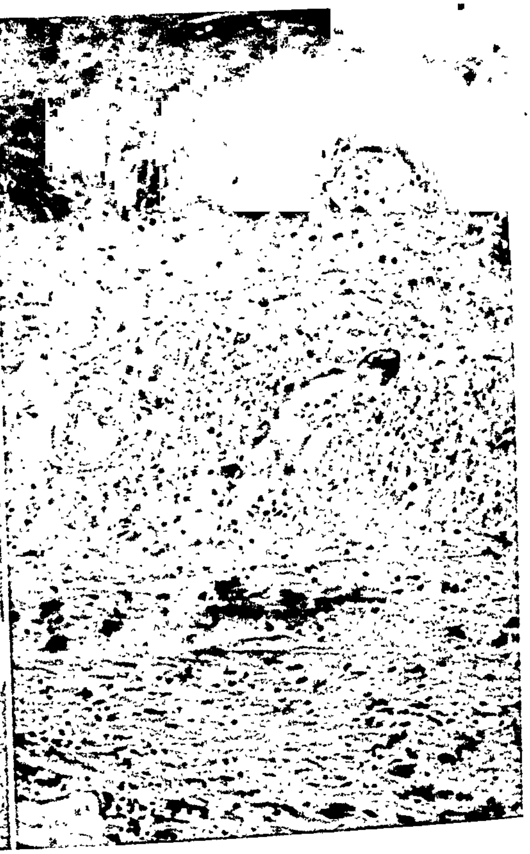

Fig. 9.

Fig. 8.—(A 16631) Section through margin of carinal ulcer shows hyperplastic squamous epithelium, vascular changes, necrosis, and acute inflammation. No tumor cells can be identified.

Fig. 9.—(A 16631) Extensive squamous metaplasia of tracheal mucosa with some chronic inflammation and vascular changes presumably the result of radiation.

PERICARDIAL FAT NECROSIS AN UNUSUAL ENTITY

A CASE REPORT

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A REPORT by Jackson, Clagett, and McDonald of the Mayo Foundation[1] describes 3 cases of a new disease entity, which these authors termed pericardial fat necrosis These authors stated that a review of the literature revealed no other such cases reported In view of the paucity of literature on this entity, the following case appears to be worthy of reporting Furthermore, the previous essayists on this disease entity made a postulation that obesity, which was present in all 3 of their cases, was probably a prerequisite of this disease Our case would indicate that this is not a constant prerequisite Furthermore, the cases reported from the Mayo Foundation were in the fifth, sixth, and seventh decades of life The case reported herein occurred in a patient in the third decade of life

CASE REPORT

A 23 year old white man was admitted to a nearby hospital on February 23, 1957, with the chief complaint of pain in the left chest The onset occurred 24 hours prior to admission at which time he was seen by his family physician He described the pain as severe precordial pain radiating into the neck and left shoulder He was pale and appeared clinically to be in mild shock It was the opinion of the physician at that time that, despite his age, a coronary occlusion was probable The patient was slightly dyspneic, but not cyanotic, when admitted to the hospital Physical examination at the time of this hospital admission was not remarkable He described pain over the left anterior chest wall and slightly less pain and tenderness over the left upper quadrant of the abdomen The blood pressure was 124/78 mm Hg The pulse was slightly rapid, but the rhythm was regular There were no abnormal heart sounds An electrocardiogram was taken on the day of admission and revealed a normal record

The laboratory findings upon admission revealed a normal urinalysis The hemoglobin and red blood count were within normal limits The white blood count was 12,250 with 54 per cent segmented cells, 17 band cells, 24 lymphocytes, 4 monocytes, and 1 eosinophil The pain gradually increased Twenty four hours after admission he was seen by a medical consultant The physical findings were essentially the same as on admission It was the opinion of the medical consultant at that time that the patient probably had epidemic pleurodynia A roentgenogram of the chest was ordered Because of the patient's acutely ill condition, this was a bedside roentgenogram On this examination a density was noted in the left lower lung It was noted by the radiologist that the shadow obscured visualization of the left heart border and the left diaphragm A follow up roentgenogram was advised and this was taken 2 days later (Figs 1 and 2) On this film the definition was much better than on the first study A round, discrete shadow was described in the left anterior base Fluoroscopy was carried out at this examination and the radiologist noted that the mass could not be separated from the cardiac silhouette During the next few days, on antibiotic therapy and analgesics, the patient's symptoms improved and he was discharged on March 2, 1957

Because of persistence of the anterior chest pain he was admitted on March 19, 1957, to Aultman Hospital On admission he was afebrile The white blood count was 7,300 cells

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of treatment well. Lacking evidence of recurrence, should such a patient be re-treated on the basis of the established experience that tumor cells almost assuredly persist?

SUMMARY

We have presented a patient who received 15,200 r to the carina over a 3-year period. His death was due to hemorrhage, secondary to slough of necrotic tissue in which no tumor was found. Our experience in this case has encouraged us to believe that radiation therapy is of value in the management of postoperative bronchial stump recurrence. In addition, our experience has shown that, in selected cases, a second course of radiation therapy can be tolerated and should be investigated further in situations other than bronchial stump recurrence as to its potential use in the long-term control of bronchogenic carcinoma.

We would like to thank Dr. Roger Terry, Associate Professor of Pathology, and Dr. Marion Emerson, Instructor in Medicine, both of the University of Rochester School of Medicine and Dentistry, for their guidance and assistance in preparing this paper.

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with a normal differential A roentgenogram of the chest was taken just prior to surgical exploration This demonstrated essentially the same findings as on the roentgenogram taken at the other hospital soon after his admission there On March 20, 1957, an exploratory thoracotomy was performed through a classical left posterolateral approach An indurated mass, measuring 7 0 by 4 5 by 2 0 cm, was found attached to the pericardium near the apex of the heart It was adherent to the anterior chest wall and the lingula of the lung by fibrinous adhesions The areas of adhesions showed acute inflammatory changes

The mass itself was attached to the pericardial surface by a broad flat surface, measuring 7 0 by 4 5 cm It was coarsely lobulated and was light brown in color Grossly, it resembled an adrenal gland This mass was dissected from the anterior chest wall, lingula, and the pericardium The bleeding points were ligated and the chest was closed with drainage in the routine manner The patient was discharged on March 27, 1957, on the ninth postoperative day His postoperative course was uneventful and he was very promptly relieved of his precordial chest pain It should be noted here that the patient was 5 feet 10½ inches tall, and weighed 155 pounds

The microscopic examination of the specimen is as follows* The specimen is composed of fat cells, many of which show granular degeneration or necrosis In places the cell membranes are indistinct or are ruptured so that small pseudocysts are formed No granulomatous reaction is present The fat is divided into lobules by delicate strands of connective tissue which show lymphocytic infiltration At the periphery of the specimen there are numerous groups of xanthoma cells, associated with mild fibroblastic proliferation and exudation of lymphocytes (Fig 3)

COMMENT

We believe this is the same clinicopathologic entity described by Jackson, Clagett, and McDonald However, we would point out that our patient was several years younger than any of the 3 patients reported by these authors, and, in contradistinction to the obese patients in which they found this disease, our patient was a rather thin person The previous authors attempted to relate the etiology of this condition to a similar condition found in the appendices epiploicae and also in the omentum Our case has not elucidated the pathogenesis of this unusual and apparently rare condition In view of the cases thus far reported, it would appear that surgical excision of the necrotic, inflamed mass gives prompt relief However, this may not prove to be the ideal therapeutic approach if more is learned about this disease

SUMMARY

1 A case of fat necrosis involving the parietal pericardial fat is reported

2 To the best of the author's knowledge, this is only the fourth such case to be reported in the literature

3 The outstanding clinical features of this condition are acute anterior chest pain, resembling the pain of coronary occlusion On roentgenograms there is an area of increased density adjacent to the left border of the pericardium

4 Histologically, the lesion resembles fat necrosis found in other situations

5 The etiology of this condition remains obscure

REFERENCE

1 Jackson, Robert C, Clagett, O Theron, and McDonald, John R Pericardial Fat Necrosis, J THORACIC SURG 33 723, 1957

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*The microscopic slides were reviewed and described by Dr Simon Koletsky, Professor of Pathology Western Reserve Medical School Cleveland Ohio

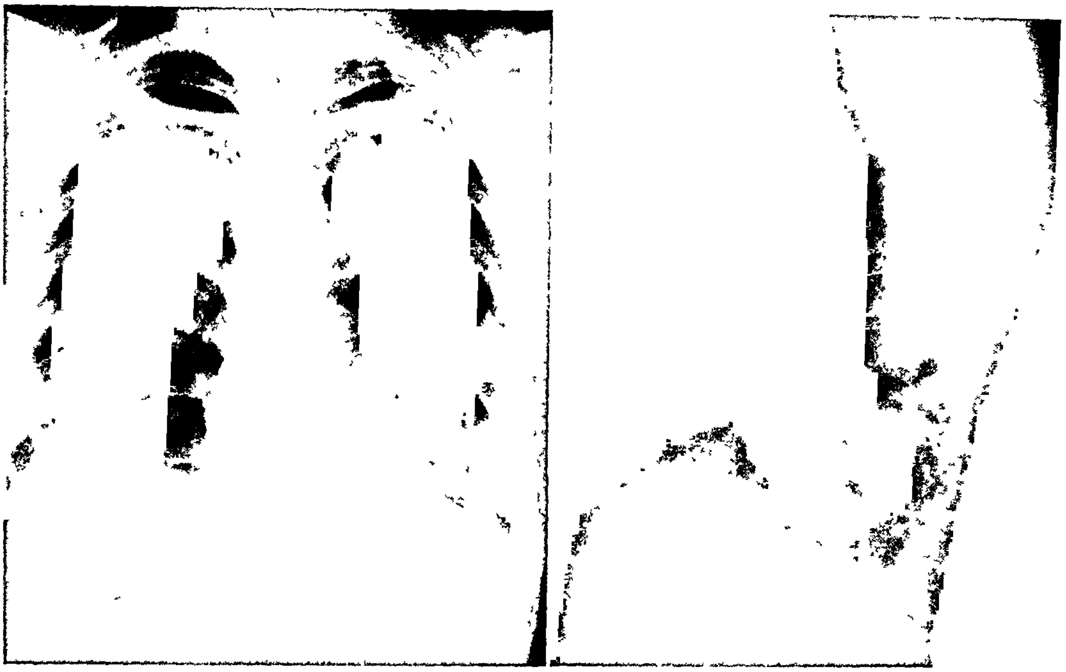

Fig. 1. Fig. 2.

Figs 1 and 2.—Posteroanterior and left lateral roentgenograms of the chest demonstrating density near the apex of the heart due to pericardial fat necrosis.

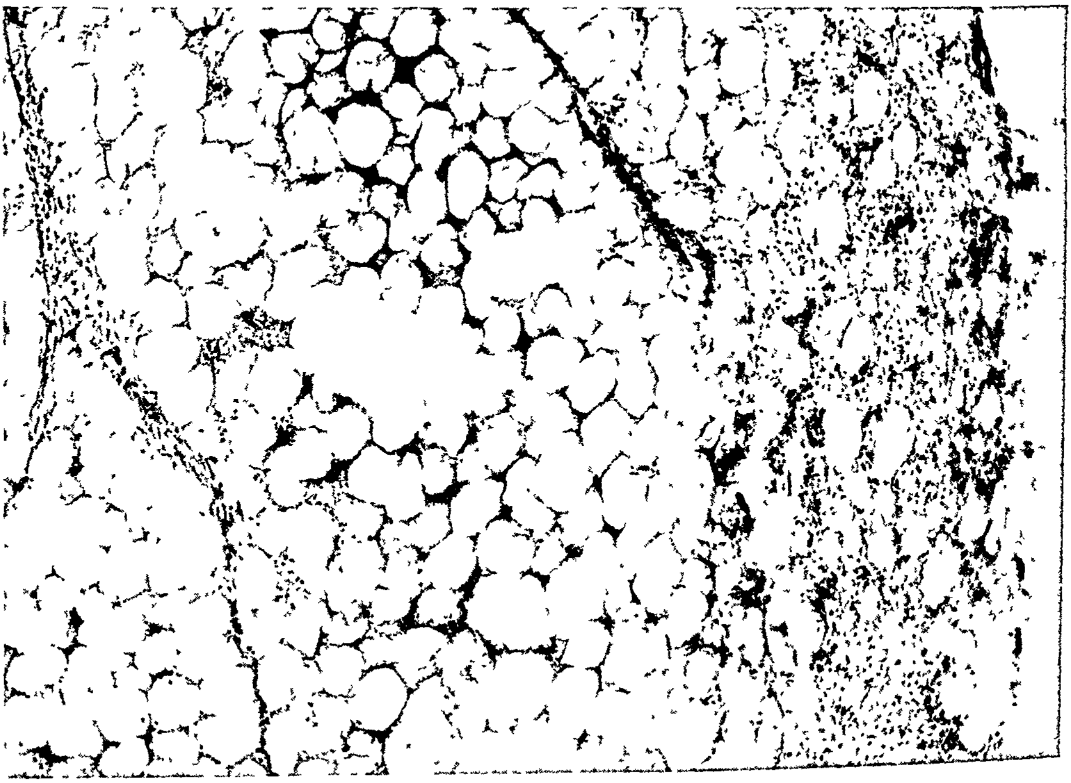

Fig. 3.—Necrosis of fat with formation of pseudocysts. Xanthoma cells are present at the periphery. (Hematoxylin and eosin; ×109.)

since forceful respiration and periods of apnea (Valsalva exercise) may lead to poor and thus unsatisfactory filling of the veins. One roentgenogram is usually all that is required as long as the film is exposed *during* the injection of the last 1 to 2 c.c. of contrast medium. Delayed exposures do indeed reveal only a minute residual deposit of contrast substance within the medullary cavity of the sternum. If the films are found to yield satisfactory information, the needle may be withdrawn and the patient sent back to the floor. No special aftercare is required. Internal mammary venography was in several instances performed on ambulatory patients who, after completion of the procedure, were discharged home without incidence.

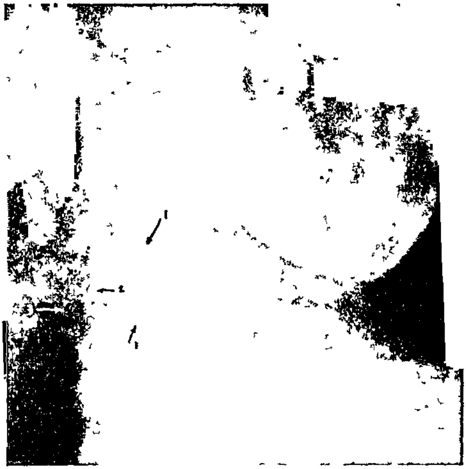

Fig 1.—*Normal* internal mammary vein pattern. Case of cystic teratoma of the anterior mediastinum *1*, Small veins draining the medullary cavity of the sternum and continuing in longitudinal direction, *2 3*, Less densely filled portion of internal mammary vein.

DISCUSSION

Internal mammary venography was carried out in a series of 50 unselected patients with primary breast disorders of benign and malignant nature. Included also were patients with postoperative metastatic carcinoma to axillary and supraclavicular nodes, and with or without evidence of involvement of the chest wall.

Although the limited number of patients examined by this method does not allow statistical conclusions, there are nevertheless several observations worthy of comment.

The injected contrast medium readily escapes, under normal conditions, the medullary cavity through several small veins which join a rather large venous channel on each side of the sternum (Figs. 1 and 2). Th [illegible] ow a

INTERNAL MAMMARY VENOGRAPHY

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THE value of intra-osseous venography in the demonstration of certain venous pathways, which hitherto partly or completely escaped roentgenographic visualization by conventional means, is now firmly established and confirmed by numerous reports. In the following report some aspects of this method will be discussed.

TECHNIQUE

The usual tests concerning possible idiosyncrasies to either the contrast substance or the local anesthetic agent must be taken. Full cooperation by the patient is ensured by explaining the procedure and mentioning the moderate amount of local discomfort experienced during the injection. Adequate sedation is obtained with meperidine hydrochloride (75 to 100 mg.) and secobarbital sodium (0.1 gram). Both drugs are administered one hour prior to the procedure.

The patient is supine on the x-ray table with both arms elevated and the correct positioning is verified with a scout film. A cutaneous wheal is raised with 1 per cent procaine hydrochloride in the midline of the sternum at the level of the fourth rib anteriorly. Additional infiltration of the sternal periosteum is advisable. A 16-gauge Rosenthal bone marrow aspiration needle is then introduced with a rotary motion into the medullary cavity of the sternum. The needle should be inclined approximately 30 degrees so as to direct its tip cephalad. The aspiration of a small amount of bone marrow as well as the accompanying sensation of local "pressure" speak in favor of adequate placement of the needle.

The radiopaque substance used in the present study was a 50 per cent solution of Hypaque† (3,5 diacetamido-2,4,6 triiodobenzoate) which, in contradistinction to other previously employed media, was found to exhibit a remarkably low incidence of ill effects, to be much less painful to the patient, and to yield roentgenograms of better contrast. An ordinary 10 c.c. manual syringe is connected to the needle by means of a piece of plastic tubing. The injection of contrast material (8 to 10 c.c.) is carried out under constant and moderate pressure. The patient is invited to breathe regularly through the open mouth

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†Hypaque was made available through the Research Department of Winthrop-Stearns, Inc.

Since the internal mammary vessels are normally maintained in a more or less fixed position, frank displacement of these veins by retro or parasternal masses will rarely occur. There will rather be an encroachment upon the wall of these vessels with resulting narrowing of their lumen. In more advanced cases (Fig 2), there may be complete block of the flow of blood within the internal mammary vein at the level of the lesion with accompanying development of collateral venous channels. Some degree of marrow stasis due to delayed outflow from the sternal marrow cavity may also be observed and is a prominent feature in cases of bilateral internal mammary vein obstruction. The collateral channels may communicate again with the involved vein at a level above the obstruction or with intercostal veins and even with the internal mammary vein on the opposite side. Furthermore, there is increased filling with contrast medium of the distal portion of the internal mammary veins.

The histologic evaluation of material removed by open biopsy from the site of demonstrable or suspected obstruction leads to the conclusion that various disorders may partially or completely block the internal mammary vein at any given level. Such lesions consisted of prominent parasternal fat pads, fibrous plaques following external irradiation, abnormally narrow vein segments, and metastatic cancer involving the internal mammary nodes.

In other words, internal mammary vein obstruction per se does not speak in favor of carcinomatous involvement of internal mammary lymph nodes even if the presence of cancer, mostly of the breast, has definitely been established. Conversely, the demonstration of a partial or complete block of the internal mammary vein in patients who are clinically "cured" from their malignant process may indicate the existence of a hidden and unrecognized focus of metastatic carcinoma. What, furthermore, can be learned from these observations is that internal mammary venography enables the surgeon to biopsy the correct site of possible metastatic involvement, be it prior to surgery or after treatment. Hence, not only a better selection but also a more adequate follow up of certain patients should be possible with the initiation of proper secondary therapy at an earlier and thus more favorable time.

It is doubtful whether internal mammary venography would be of any benefit in the study of certain anterior mediastinal tumors when their location, relationship to the surrounding structures, and other characteristics are concerned. It can be anticipated that most lesions in that particular location will not encroach upon the internal mammary veins (Fig 1).

SUMMARY

The technique of internal mammary venography is described and the advantages as well as limitations of this procedure are emphasized.

The more frequent use of this method in the follow up study of clinically "cured" patients may well lead to a better evaluation of the therapeutic results in breast cancer.

My sincere gratitude goes to Dr H C Moss, Associate Chief, Breast and Soft Tissue Department, for his much appreciated clinical cooperation as well as to Dr F P Lessmann for making available the facilities of the Department of Diagnostic Radiology.

course parallel to the sternal edge and their anatomic location corresponds to the one occupied by the internal mammary veins. The caliber of these veins remains more or less equal and filling in proximal direction can usually be followed up to their point of entry into the distal portion of the innominate veins, while the visualization in distal direction may be variable.

The small branches which are seen to drain the medullary cavity of the sternum may not always immediately join the internal mammary veins but are occasionally seen to run in a longitudinal direction along the sternal border. It is important to recognize this fact and not to mistake these veins for narrowed segments of the internal mammary veins, especially if less dense filling of these latter structures has been obtained (Fig. 1). There may, on the other hand, exist a real narrowing of the internal mammary vein itself, independent of any extrinsic pressure, which, as observed in several of our cases, usually occurs in the region of the third costal cartilage and mostly on the right. That such narrowing does not merely represent a roentgenographic artifact, due to insufficient filling of the vein with contrast medium, has repeatedly been confirmed by surgical exploration of the respective area.

Fig. 2.—*Abnormal* internal mammary vein pattern. *A*, Area of obstruction caused by enlarged metastatic lymph nodes in the right second intercostal space. Deviation of blood flow over collaterals to ipsilateral, *B* and opposite vein, *C*, *D*. Left internal mammary vein essentially normal.

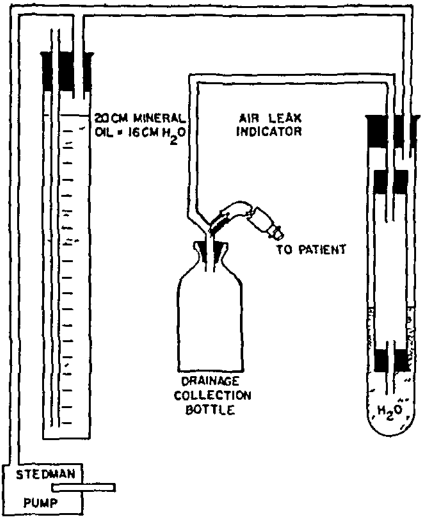
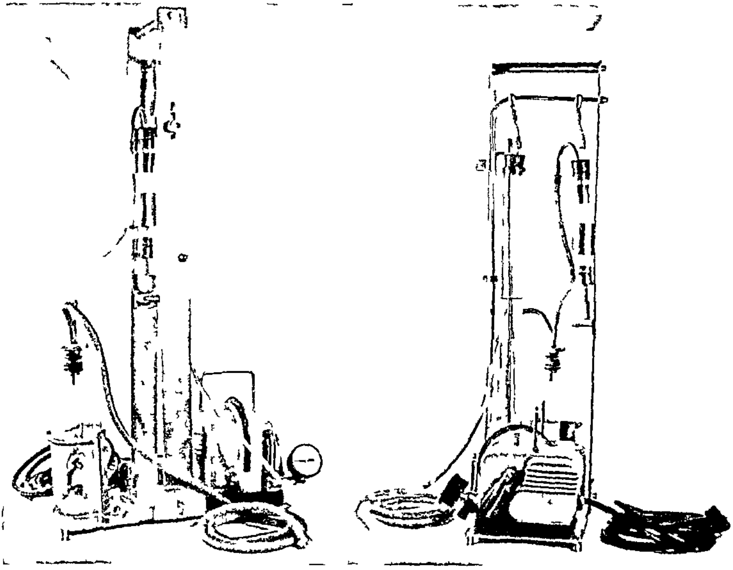


Fig 1—A diagram of the suction apparatus On the left is the Stedman pump and the tube for maintenance of a constant negative pressure The drainage bottle is in the center and the air leak tube is on the right.



A *B*

Fig 2, *A* and *B*—*For legend see opposite page*

A COMPACT SUCTION APPARATUS FOR CHEST DRAINAGE

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THE 3-bottle suction apparatus for chest drainage catheters which is described in textbooks of thoracic surgery[1, 2, 3] is a simple, effective apparatus, but it has a number of defects. The three bottles, plus a suction pump, occupy a considerable amount of space and they are difficult to move. Nurses find it troublesome to empty the drainage bottle but measurement of the fluid drainage will not be accurate unless the bottle is emptied. The connecting rubber tubes must be watched carefully to avoid kinking and obstruction. The bottles are easily tipped over and broken, thus connecting the pleural space with atmospheric air. To correct some of these defects and to make the whole apparatus more convenient for the nursing staff, we have modified the 3-bottle suction system into a compact, rugged unit of simple design.†

The three bottles of the unit—the drainage bottle, the air leak indicator, and the vacuum regulator bottle—are all small and arranged compactly on a substantial metal platform which has room for one Stedman pump (Figs. 1 and 2). The entire apparatus stands conveniently under the hospital bed. The Stedman pump maintains a negative pressure, the degree of which is regulated by the depth of the vent tube in the column of mineral oil. For a small volumn tube, such as this one, mineral oil has been better than water because it does not evaporate. If there is an air leak from the lung, this is indicated by bubbling from the small tube of the air leak indicator and this indicator bottle also serves as a water seal should suction fail. Adjustments in the amount of negative pressure can be made easily by changing either the level of the oil or the depth of the tube beneath the oil. The use of a larger inside tube in the air leak indicator prevents water from being sucked over into the drainage bottle when the patient makes a strong inspiratory effort.

All parts of the apparatus are easily disassembled for cleaning or replacement. The metal parts are stainless steel. The apparatus is designed so that only short, relatively straight, rubber interconnections are used and thus the

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†Manfactured by Oliver M. Moe, 1926 Hartford Ave., St. Paul, Minn.

Fig. 2.—*A*, Photograph of the apparatus viewed from the side. The drainage bottle is at the left, air leak indicator in the center, and the Stedman pump on the right. The tube which is attached to the patient's chest catheters is coiled at the bottom of the picture.

B, A photograph of the apparatus from the front. The Stedman pump is seen from the front and the drainage bottle is just behind it. The pressure regulating tube is on the left and the air leak tube on the right. The crossbar at the top is a handle for the apparatus.

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Original Communications

AN EVALUATION OF IMPLANTATION OF THE INTERNAL MAMMARY ARTERY INTO THE MYOCARDIUM OF PIGS AND DOGS

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SURGICAL efforts to introduce a new blood supply into the myocardium have been numerous and varied. The internal mammary artery implant as first described by Vineberg[15] in 1946 seemed a simple but intriguing approach to the problem. The reported observation that an open vessel implanted into myocardium remained open appeared to be a biologic phenomenon of great importance and deserving of intensive investigation. Consequently, when we became interested in the problem of myocardial revascularization, we were particularly interested in the procedure of internal mammary artery implant. Knowing the difficulty of evaluating any of these procedures in the human being, we began our investigation in the laboratory animal, using the weanling pig as our experimental animal.

The pig was selected primarily because its coronary circulation is of the end-artery type and very closely approximates that of the human being.[3] It has been shown that ligation of the left anterior descending (LAD) artery in the pig results in almost 100 per cent mortality within 15 minutes.[3] Logically, a highly consistent test ligation of the LAD artery might prove to be a valuable yardstick for determining the efficacy of any operative procedure in improving the coronary blood supply.

A total of 25 pigs survived the implantation of the left internal mammary artery into the myocardium of the left ventricle. Eleven of these were operated upon after a 6- to 12-week period and a test ligation of the LAD artery was

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danger of kinking is eliminated. The drainage bottle is a standard pharmacist's bottle with graduations on the side which make volume measurements simple and direct. A special stopper makes it possible to empty the bottle quickly and easily. This stopper consists of a rubber cork which is compressed in order to tighten it in the bottle neck by simply turning the knurled top of the stopper.

The apparatus has been used in the postoperative care of pulmonary and cardiac surgery patients and it has proved to be superior to the usual 3-bottle suction apparatus.

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Animals surviving the procedure were sacrificed 2 to 3 months later. Flow studies were performed through the isolated implant in all animals not having obvious gross occlusion of the implanted vessel. Normal saline solution at 120 mm. mercury pressure was used in the flow studies. Whenever an apparent flow through the implant was obtained, the saline was colored with methylene blue to locate leaks and to look for myocardial staining. Schlesinger's[13] mass was injected into every implant with a demonstrable flow. All such specimens were sectioned and studied microscopically along with representative specimens of grossly occluded implants with no flow.*

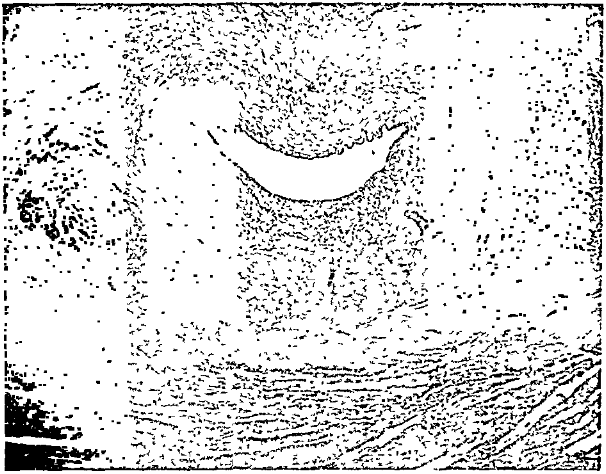

Fig 1—*Dog No. 116.* Photomicrograph of the LAD artery distal to a partially occluding ligature producing myocardial need. The lumen is still patent, although narrowed. There is minimal intimal thickening.

RESULTS

Nineteen of 23 dogs survived the operative procedure over the experimental period. All 4 mortalities occurred within 2 weeks of operation and were the result of the partial occlusion of the LAD artery. One implant of the remaining 19 was demonstrably patent by all criteria (Dog. No. 116). There was a flow of 6.5 c.c. saline per minute at 120 mm. mercury pressure in the living animal. The vessel was grossly and microscopically patent. Schlesinger's mass injected into the implant was seen to enter the LAD artery and its branches (Fig. 2). In this one animal, a significant anastomosis developed between the implanted internal mammary artery and the left coronary artery system. The other 18 implants were occluded, giving a 5.3 per cent rate of successful internal mammary artery implants.

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*We gratefully acknowledge the assistance of Drs Tobias Weinberg, Robert Solomon, and Alma Kelly of the Department of Pathology in the preparation and interpretation of this material.

performed. Of this group, 10 died promptly of ventricular fibrillation, suggesting a failure of the implant to protect the myocardium from the effects of LAD artery ligation. Post-mortem examination in each of these 10 animals revealed gross and microscopic occlusion of the implanted internal mammary artery. Fourteen of the pigs with internal mammary implants grew too large to handle for secondary operation in our laboratory. These were slaughtered at an abattoir and the hearts removed for examination. In no instance was an anastomosis demonstrable between the internal mammary implant and the coronary circulation. Only one pig in the series of 25 (4 per cent) could be considered to have a successful internal mammary implant. This animal survived the test procedure of LAD ligation and gross anastomosis between the patent internal mammary and branches of the LAD artery was demonstrable by injection of colored vinyl plastic.

It was apparent that this series was inconclusive. In using a different experimental animal, the conditions of Vineberg's experiments were not reproduced. The pigs were all young animals and this might properly be considered an unfavorable feature in the test of the procedure. The weanling pigs responded to open chest surgery with an extensive dense fibroblastic reaction. Some of the adhesions at the operative site were almost cartilaginous in consistency. This was equally true when a midsternal extrapleural approach was perfected. Another serious objection to the validity of the results was the failure to produce myocardial need. In addition, it was noted that Vineberg's results *improved as he and his associates gained technical proficiency. It seemed reasonable* that we, too, might expect a higher number of "takes" as our familiarity with the procedure grew. Accordingly, a new series was begun in dogs, duplicating the conditions of Vineberg's experiments in every detail.[15]

PROCEDURE

Healthy mongrel dogs weighing 25 to 45 pounds were operated upon through the left chest under intravenous Nembutal anesthesia. Pulmonary ventilation was maintained by positive pressure oxygen administered by a modified Emerson* resuscitator through an intratracheal tube. Myocardial need was created by partially ligating the LAD branch of the left coronary artery over a No. 23-gauge needle (Fig. 1). The left internal mammary artery was carefully dissected free between the fourth and sixth interspaces. The distal end was divided and ligated preserving a suitable intercostal branch. The myocardial tunnel in the left ventricle was made with a small curved clamp and planned to prevent the possibility of angulation of the internal mammary as it was pulled through. Just prior to burying the implant, the selected intercostal branch was divided flush with the artery so that there was free and vigorous bleeding into the tunnel. The distal end of the implant was then anchored to the exit of the tunnel by a suture-ligature. The pericardial fat pad was utilized to prevent adherence of the internal mammary to the denuded sternum rather than as an additional source of blood to the myocardial surface.

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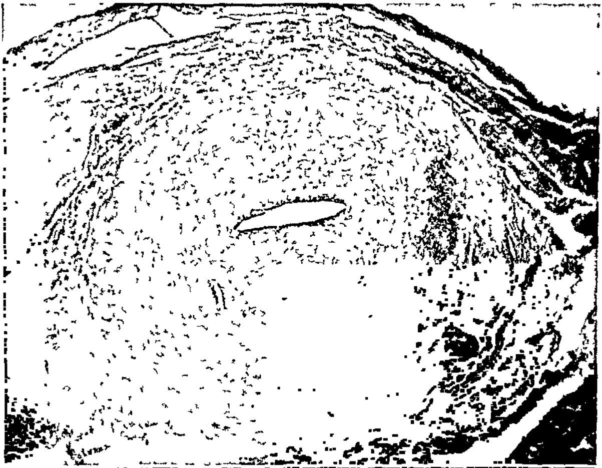
*J. H. Emerson Company, Cambridge, Mass.

Fig 4



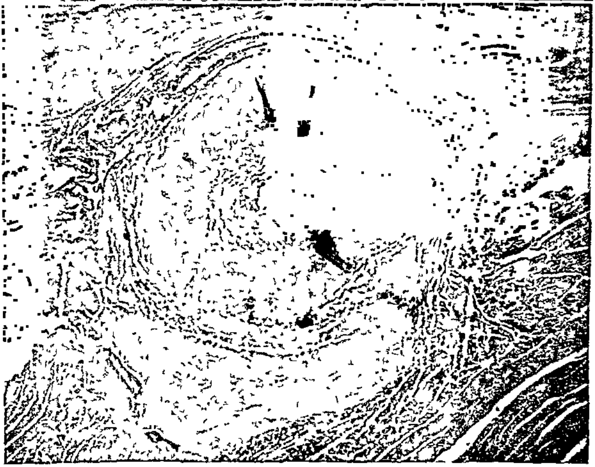
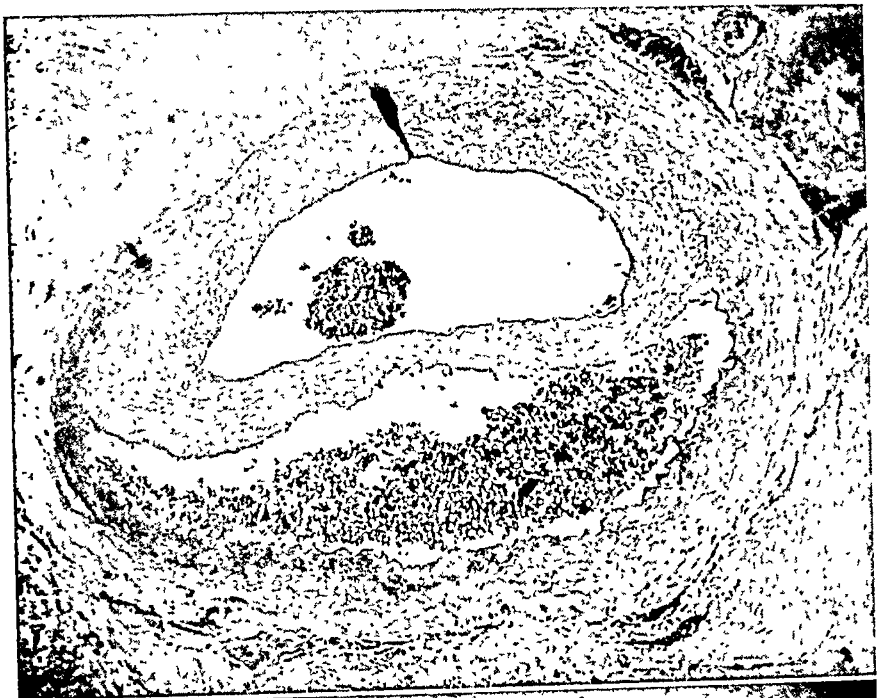


Fig 5

Fig 4 —*Dog No 208* Section through the internal mammary artery proximal to the myocardial tunnel Note the extensive intimal proliferation resulting in marked narrowing of the lumen

Fig 5 —*Dog No 167* Section through the internal mammary artery within the myocardial tunnel Note the extensive intimal proliferation and marked narrowing of the lumen There is Schlesinger's mass present within the lumen of the implant but none demonstrable in the small branches nearby

Fig. 2.



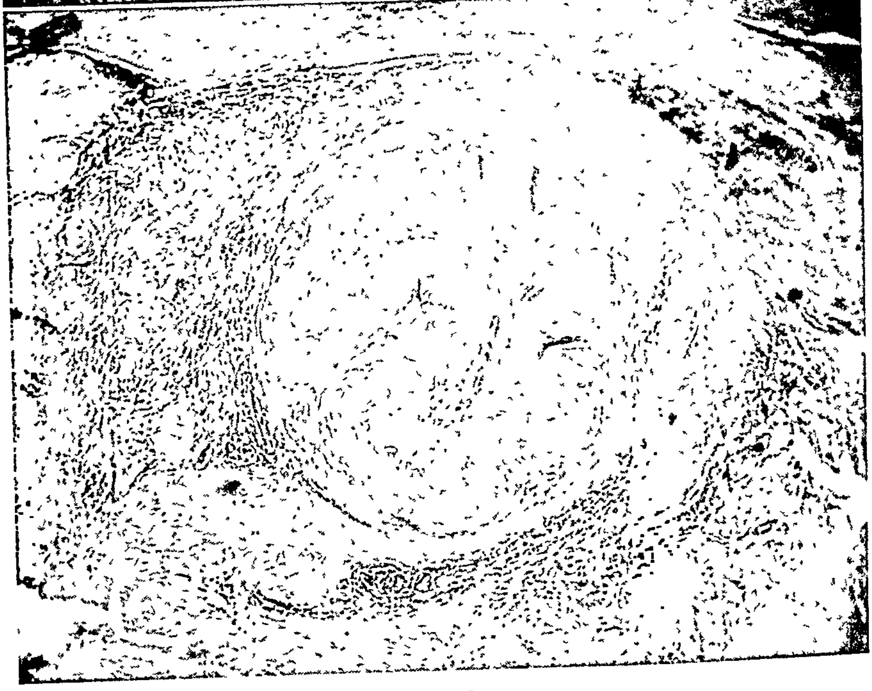


Fig. 3.

Fig. 2.—*Dog No. 116.* Section through the myocardial tunnel demonstrating a widely patent implanted internal mammary artery. There is slight intimal thickening with dissection of the intima from the media. Whether or not this is an artefact is not known. Note the Schlesinger's mass present in both the true lumen and false passage as well as within the small branches nearby.

Fig. 3.—*Dog No. 143.* Section through the internal mammary artery within the myocardial tunnel. In spite of an apparently successful flow study, the lumen is completely obliterated. The obliteration is due to compression of the vessel wall as well as intimal proliferation.

SUMMARY

1. Implantation of the internal mammary artery into the myocardium was not a satisfactory revascularization procedure in our hands.

2. A significant anastomosis was demonstrable in only one of 19 dogs (5.3 per cent) subjected to implantation of the internal mammary artery after partial ligation of the left anterior descending artery.

3 Successful anastomosis was demonstrable in only one of 25 pigs (4 per cent) subjected to a similar procedure.

(For References, see page 717)

One animal (Dog No. 143), in which a 3.0 c.c. per minute flow was obtained, proved to have an occluded implant on subsequent Schlesinger's injection and microscopic study (Fig. 3). This apparent discrepancy proved to be due to minute leaks in the internal mammary artery prior to its entrance into the myocardial tunnel. The addition of methylene blue to the perfusate eliminated this source of error in subsequent experiments.

DISCUSSION

It is difficult to evaluate objectively the efficacy of a revascularization procedure on the human myocardium. Obvious clinical improvement may follow an operative procedure but may be entirely unrelated to the specific operation employed. The precise evaluation of an advocated operative procedure by suitable laboratory study is of primary importance.

In our hands, the implantation of the internal mammary artery into the myocardium was not a satisfactory method for improving the blood supply to the heart. An occasional successful result was obtained, but not with sufficient frequency to warrant our employment of this procedure in patients. Although other investigators have reported similar findings, their evidence was inconclusive. Glenn and Beal[6] reported results in 6 dogs observed for a period of 1 to 6 weeks. Their operative procedure differed considerably from that advocated by Vineberg and their adverse findings must be regarded as inconclusive. Bakst and co-workers[1] reported poor results with implantation of the internal mammary artery but used only 9 animals in none of which was myocardial need established. In our study, the usual criticisms were obviated. The size of the series was adequate. The technique employed was exactly as described by Vineberg. Myocardial need was established. The operating surgeons had previous experience in internal mammary implants and were not attempting a new procedure for the first time.

The often-described phenomenon of intimal proliferation was consistently present (Figs. 4 and 5) and could be found to some degree even in the successful implant. This may eventually be circumvented by the use of a larger vessel,[5, 10, 12] or an artificial graft.[14]

In performing a flow study on an implanted vessel, it is essential to ascertain exactly where the perfusate is going. An anastomosis via adhesions to the sternum or mediastinal fat may result in a misleading flow study. Small leaks from such vessels which have been divided are readily detected when the perfusate is colored with methylene blue. Failure of the dye to discolor the myocardium is additional evidence that flow through the implant is not synonymous with flow into the heart muscle.

Injection of the implant with Schlesinger's mass is important in establishing the caliber of the anastomotic channels. The mass will not traverse a vessel with a diameter less than 40 micra.[13] Only Dog No. 116 was successfully injected with this material.

Although these first attempts to introduce a new blood supply into the insufficient myocardium have been disappointing, investigation of the value of certain modifications of the original procedure are in progress in this laboratory.

results revealed that in 25 young pigs and in 19 dogs, the lumen of the implanted internal mammary artery became obliterated within 4 weeks in all but one animal of each species[11] The present paper presents the experiments which were carried out in an effort to understand the cause of the intimal proliferation and the consequent obliteration of the artery which had been implanted into the myocardium It will be shown that when continuous flow through the implanted artery is maintained by plunging the open bleeding end of the vessel into the lumen of the right ventricular cavity, the implanted vessels do not develop intimal proliferation

EXPERIMENTAL STUDIES

Control Group—The use of an artery larger than the internal mammary was suggested by the notion that if the lumen were narrowed to 50 per cent or even 75 per cent of its original size, an adequate flow of blood still might be supplied to the heart muscle This thought was also present in the minds of other investigators, who have since presented the results of their work[5, 10] Consequently, the left carotid artery was mobilized through a vertical cervical incision, 2 cm to the left of the midline The left chest was then entered through the fourth intercostal space and, with blunt dissection from within the thorax and the neck, the transected carotid artery was freed sufficiently to bring it into the pleural cavity The pericardium was incised and after one tangential opening, 4 mm in diameter, was made in the carotid artery, the vessel was pulled into a tunnel in the left ventricular myocardium An inspection was made to be certain that the artery was not significantly kinked at its origin as it was directed downward toward the heart, and that it was not obstructed at the point of entry into the myocardium The pericardium was loosely reapproximated and the chest wall closed

This operation was performed on 14 dogs which served as the control group At the end of one month the animals were sacrificed, and the hearts with their implanted vessels were studied microscopically In 13 of the 14 dogs, the lumen of the implanted vessel was reduced to 25 per cent or less of its original area This was caused by intimal proliferation in 11 of the animals, while in the 2 others a thrombus was responsible for the occlusion In one dog of this group, the portion of the vessel within the myocardium had retained 80 per cent of its original lumen, but the more proximal portion was narrowed to 20 per cent and was the site of a fresh thrombus Thus, at the end of one month, these implanted carotid arteries were unable to deliver a significant extracardiac source of blood to the myocardium

Implantation Modified to Include Placing Carotid in Right Ventricle—The high incidence of intimal proliferation and thromboses as reported by others,[1, 2, 9, 11] who used the internal mammary artery, was confirmed by our experiments in which a larger vessel was implanted by the standard operative technique Our attention was directed, therefore, to the problem of trying to understand the cause of the intimal proliferation Previous work had suggested that the maintenance of flow through the implanted vessel was important[4]

MAINTENANCE OF PATENCY OF CAROTID ARTERIES IMPLANTED INTO THE MYOCARDIUM

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IN 1946, Vineberg[15] introduced the ingenious procedure of placing the left internal mammary artery into a myocardial tunnel. His subsequent reported experiments presented evidence to indicate that such an implanted vessel remained patent and developed arterial connections with the coronary vascular system, thus furnishing an extracardiac source of blood to the myocardium.[16, 17] Not only was there an anatomic connection but, also, this extracardiac arterial blood was capable of protecting the heart muscle against both the mortal and morbid effects of coronary artery occlusion in experimental animals and in man.

This procedure has not attained widespread acceptance for, in the hands of other investigators, the results were not the same as his. Glenn and associates[7] observed that the new vessels which formed were small, resembling the capillaries seen in granulation tissue, and tended to disappear some time after the operation. Bakst[1] and Maniglia[9] and their co-workers implanted the mammary artery into the myocardium of 9 dogs and, at the time of sacrifice 6 months later, found that the lumen was obliterated to 10 per cent or less of its original size in 7 of the 9 animals, while in the remaining 2 dogs it was 30 per cent and 50 per cent patent. The physiologic studies from the same laboratory gave no indication of any benefit from the implantations. Beck[2] has also evaluated this procedure in dogs, and has been unable to find any evidence that a significant amount of extracardiac blood is delivered by the implanted vessel. On the other hand, Sabiston, Fauteux, and Blalock,[12] using a larger artery (carotid) and a somewhat different technique, were able to demonstrate that the lumens of the implanted vessels remained patent in 28 of 32 dogs. However, when they measured the amount of blood supplied to the heart muscle by the implanted vessel, the values averaged no more than 5 c.c. per minute.

Because of the contradictory nature of the reports, and because the concept of arterial implantation into the myocardium appeared to be a biologic phenomenon, sufficiently unique and important to merit careful investigation, we began to evaluate the procedure in this laboratory 3 years ago. The early

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The fourth dog was a technical failure in that the distal end of the implanted vessel had retracted into the right ventricular myocardium so that flow into the right ventricular cavity was not possible. The lumen of this implanted vessel was occluded throughout its course in the myocardial tunnel of the left ventricle resembling the arteries in the control group. These experiments appeared to support the argument that the trauma to which the implanted artery was subjected by the repeated muscular contractions did not result in intimal proliferation, as long as blood was flowing through the vessel.

Findings of Implantation With Flow Into Right Ventricle.—In 29 dogs the carotid artery was implanted into the left ventricular myocardial tunnel with a tangential opening having been made in the artery, and the distal open end of the artery was placed in the right ventricular cavity. Twenty-two of the animals were sacrificed at 1 to 3 months postoperatively, while 7 were followed for 6 to 10 months. A period of 3 to 4 weeks was considered the minimal lapse of time for evaluating the fate of the implanted arteries since, in the control group, the lumens were always obliterated by that time interval. Unexpected obstruction was noted in the distal end of the artery in 14 dogs. In 6 instances, the vessel was not placed accurately because of faulty technique, and it was found to be occluded because it had retracted into the ventricular muscle or was blocked by the papillary muscle. Occasionally (4 dogs) a satisfactory preparation showed a large polypoid thrombus which occluded the end of the artery. Such an obstruction to the distal end of the implant lying in the right ventricular cavity is shown in Fig. 2. In the other 4 dogs, the end of the artery was occluded without an obvious cause. These 14 dogs, therefore, did not have blood flowing through the implanted artery into the right ventricular cavity, and thus represented a preparation similar to the control group. They may be considered as "accidental controls." In the remaining 15 animals, the lumen of the vessel in the right ventricular cavity was either entirely open or had become narrowed during the postoperative period, but had apparently permitted continuous flow of blood through the vessel. These represent the "test animals" which can be compared with both the control and accidental control groups. The desired result of an open distal end which permits blood flow into the right ventricular cavity is shown in Fig. 3.

Results in the Accidental Control Group.—The 14 dogs in this group revealed that the lumen of that portion of the vessel passing through the left ventricular myocardium was reduced to 25 per cent or less of its original size in 11 animals, while in 3 it was 75 to 100 per cent patent. This suggests that perhaps the distal end of the vessel was delivering blood into the right ventricular cavity for some period of time during the postoperative course of the latter 3 animals. However, when these findings are combined with the control group of 14 dogs, there were only 3 dogs among the 28 (11 per cent) in whom the lumen of the implanted arteries remained patent one month after operation. These results were obtained in all those animals with an implanted carotid artery, which had one good-sized tangential opening in the implanted portion of the vessel and which were occluded distal to the tunne

This was shown by the procedure of dividing the internal mammary artery proximally after it had been freed from the chest wall, carrying the artery through a ventricular tunnel, and anastomosing the proximal end of the vessel to the left atrium. By such a technique Carey and associates[4] were able to demonstrate a marked reduction in the occurrence of intimal proliferation as compared with the control series. The procedure which we instituted was to perform the same operation as described in the control group above, but to place the distal end of the carotid artery with its open end into the right ventricular cavity. This procedure is illustrated in Fig. 1.

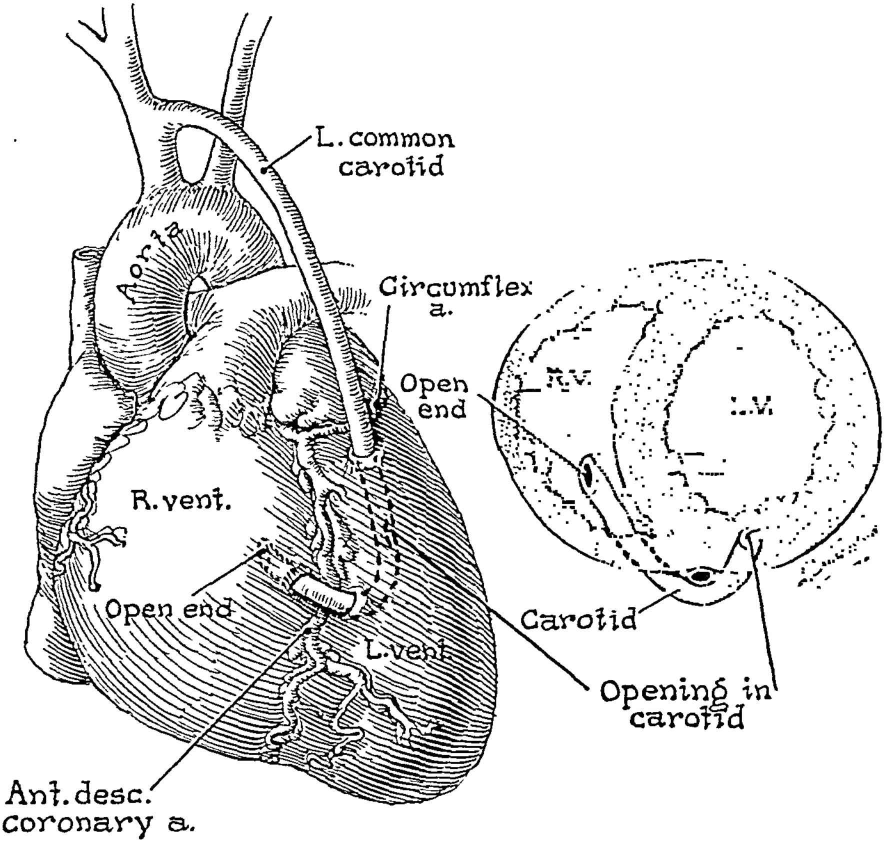

Fig. 1.—Illustration of the double implant type of operation where the carotid artery with one tangential opening is implanted into the left ventricular myocardium and the distal open end of the vessel is placed in the right ventricle.

Effect of Cardiac Contraction on the Implanted Vessel.—It seemed advisable to determine whether the trauma which the artery sustained by repeated ventricular contraction was responsible for the intimal proliferation. Therefore, the procedure just described was carried out on 4 dogs with the exception that no tangential opening was made in the artery as it passed through the myocardial tunnel of the left ventricle. Thus, we could determine the effect of the cardiac trauma alone. In 3 of these 4 animals, which were sacrificed at the end of one month, the artery appeared grossly as soft and patent as the day it had been implanted. This was in striking contrast to our control group, in which the arteries were thickened, rigid, and almost completely obliterated.

TABLE I. PRESERVATIONS OF PATENCY IN THREE TYPES OF CAROTID IMPLANTATIONS

GROUPS*	TOTAL NO. OF DOGS	0-25% PATENCY NO. OF DOGS	80-100% PATENCY NO. OF DOGS
1. Controls	14	14	0
2. Accidental controls	14	11	3
3. Test	15	0	15

*The control group represent the animals which had a standard type of implantation of the carotid artery into the myocardium of the left ventricle. The accidental controls were those animals in which the distal open end of the artery was placed in the right ventricle but which was obstructed due to thrombosis or faulty technique. The test group are those dogs with a persistent open end of the implanted vessel delivering blood into the right ventricle.

into the implanted artery, the entire left coronary arterial system was seen to fill with the blue dye. This animal was sacrificed 9 months after implantation. In 11 of the 15 test preparations either a flow study or a Schlesinger's mass injection, or both, were done. The 6 flow studies performed did not reveal any significant amount of blood flow. Six of the 8 injected specimens had good anastomotic connections between the implanted vessel and the coronary arterial tree. However, flow studies done in 3 of these 6 were disappointing in that they only demonstrated 1, 3.2, and 9 c.c. per minute.

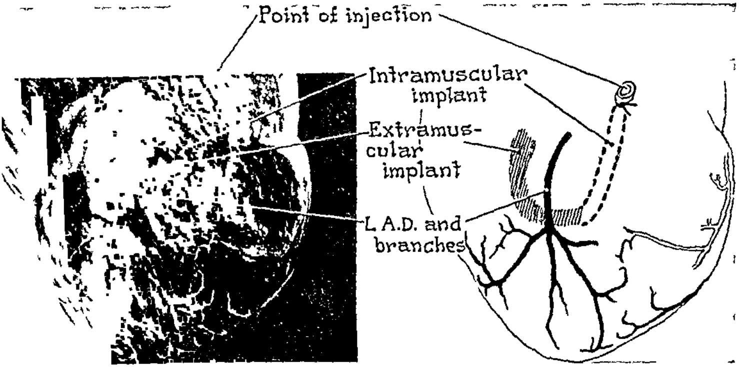

Fig. 4.—Bas-relief photograph. Schlesinger's mass has been injected into the implanted carotid artery 9 months after its placement. The blue-stained mass filled many of the branches of the anterior descending branch of the left coronary artery. On the right is an interpretive diagram, which helps to overcome the deficiencies of the black and white print. The heavy markings indicate those vessels which were seen to contain the blue mass. Similar findings were noted in 6 of the 8 dogs in which such injection studies were done. However, the presence of these connections between the implanted and the coronary arteries was not associated with a significant amount of measured blood flow.

Details of the Pathologic Findings.—An examination was made of each heart at the termination of the experiment. Gross inspections were made and blocks were taken for microscopic examination from the carotid artery proximal to the implant, from the artery within the tunnel, at a point distal to the tunnel and when the double implant was made, adjacent to the site of egress into the right ventricle. Sections were stained with hematoxylin and eosin and with a Weigert elastic-van Gieson stain where indicated.

Results in the Test Animals With Flow Into the Right Ventricle.—These 15 dogs were considered satisfactory for evaluating the effect of continual blood flow through an implanted vessel. In contrast to the findings just recorded, all of these vessels were soft and pulsatile. On microscopic examination, the lumens were found to be from 80 to 100 per cent patent. Table I summarizes the results in control and test animals. When the 3 dogs in which the effect of muscular contraction on the artery are included with this test group, it may be noted that 100 per cent of the 18 implanted arteries remained patent when they had an open distal end to allow continuous flow.

Fig. 2

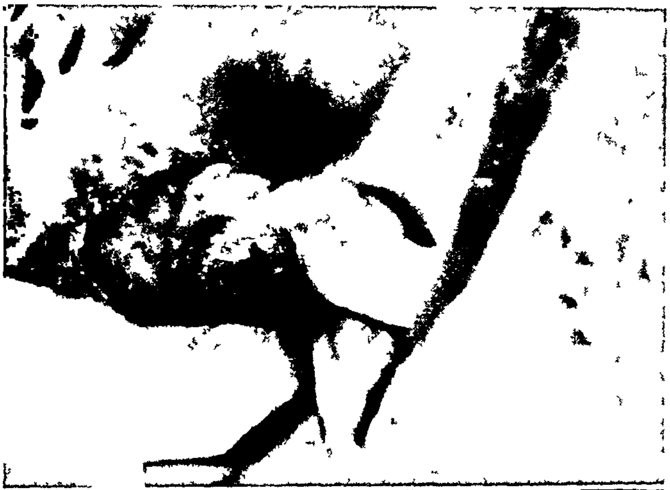

Fig. 3

Fig. 2.—Photograph showing an obstruction by a thrombus of the distal end of the implanted vessel lying in the right ventricular cavity. The external portion of the carotid is shown on the right, lying on the surface of the left ventricle. It passes through the cut myocardium of the right ventricle and is shown lying in the cavity. The attached thrombus is seen on the left. When this prevents continuous flow through the carotid artery, the implanted vessel shows marked intimal proliferation.

Fig. 3.—In this specimen, the desired result of an open distal end of artery is seen. The artery is lying in the center of the field within the right ventricular cavity just below the pulmonic valve. The implanted portion remains soft and patent, with little or no intimal proliferation.

That anastomotic connections with the coronary circulation did develop in some of these animals is shown in Fig. 4. When Schlesinger's mass was injected

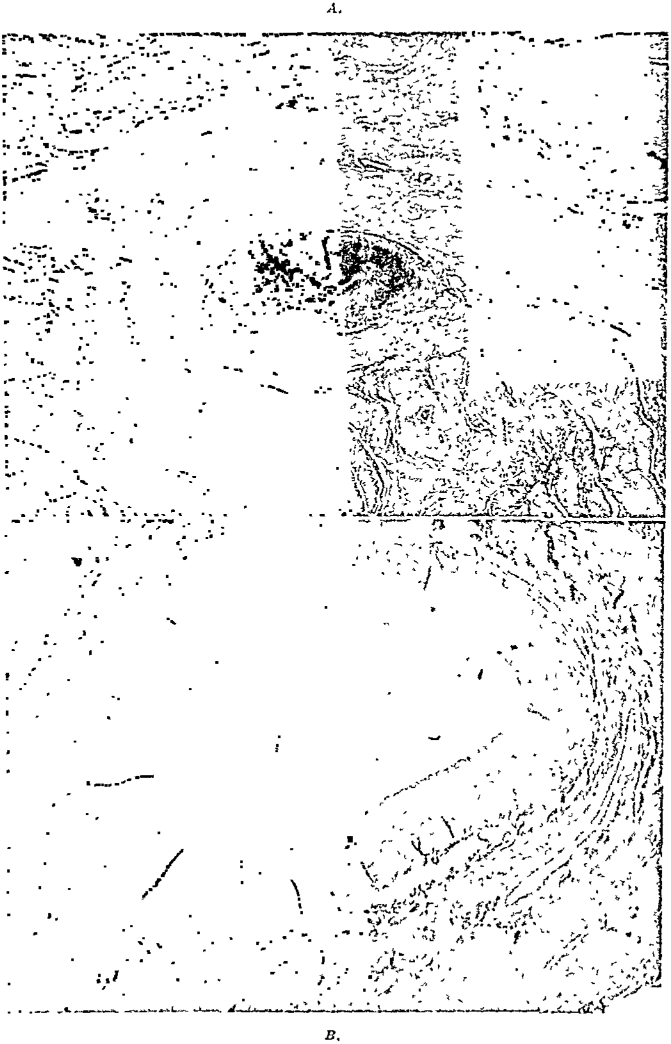

Fig. 6.—*A*, Obliterated carotid artery in a control animal with the artery lying within the intramural tunnel, 7 weeks postoperative. *B*, In contrast, the artery in the test animal with free flow into the right ventricle shows minimal intimal proliferation, 7 months postoperative. The perivascular scar formation is notably small.

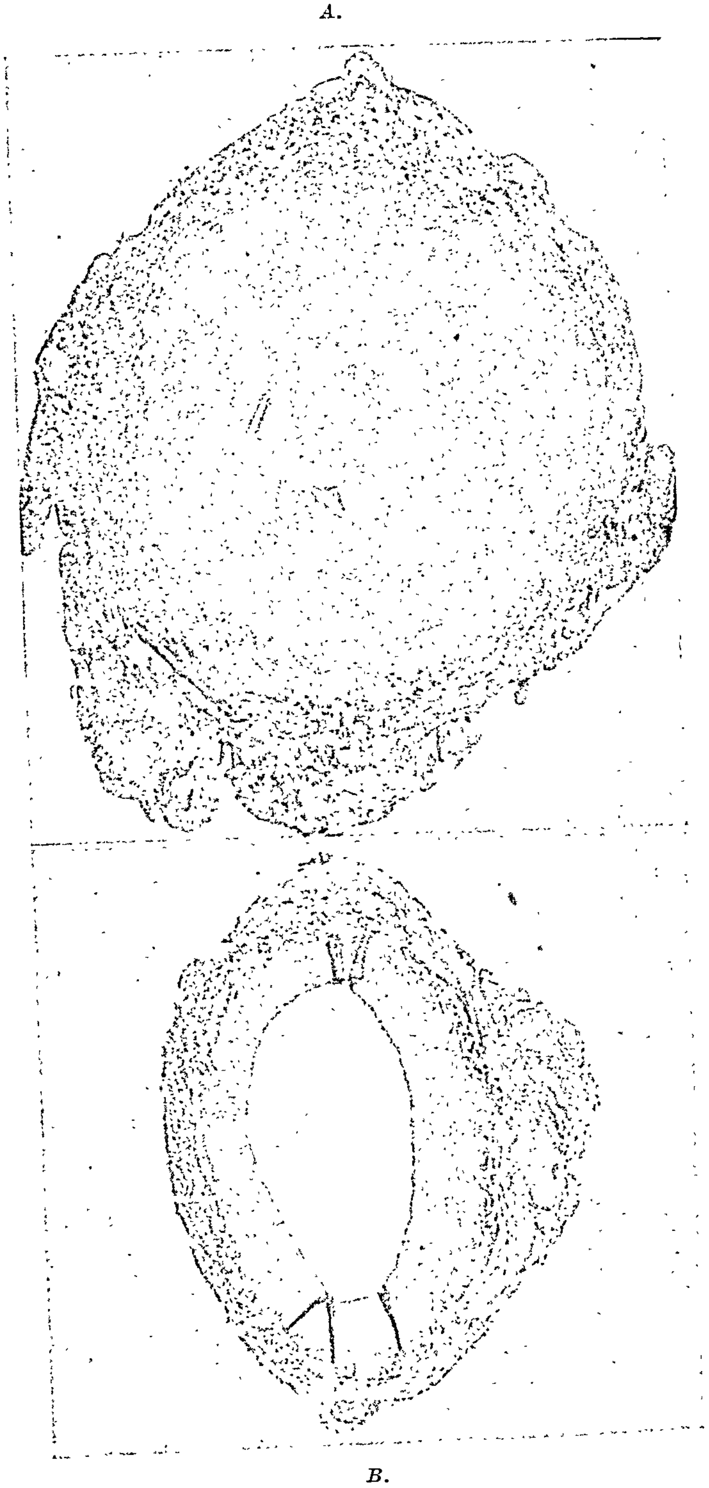

Fig. 5.—*A*, Control preparation; artery proximal to the tunnel, 7 weeks postoperative. The vessel is obliterated by intimal proliferation and organized thrombus. *B*, Test preparation with carotid terminating in right ventricle; artery proximal to myocardial tunnel, 7 months postoperative.

As others have observed, no hematomas were noted about the vessels in the myocardium even at the points where openings had been produced. There was insignificant phagocytized hemosiderin, but an occasional heart exhibited calcifications adjacent to the implant. The arteries in the tunnels were surrounded by collagenous scar tissue but this reaction, present also in the controls, was variable and did not depend on the duration of the experiment. In some instances there was surprisingly little scar tissue formed about the vessels.

Obliteration of the implants occurred in the controls and "accidental controls" by several mechanisms. Intimal proliferation was present to some extent in all instances, featured by a growth of loose textured, weblike connective tissue. This was accompanied by some splitting of the internal elastic lamella. Thrombosis occurred in one half of the cases with early or late organization depending upon the duration of the experiment. The thrombotic masses formed on the intimal elevations and also on areas of necrotic intima for, in several hearts, the walls of the carotids were necrotic from adventitia to intima. It seems highly unlikely that the recanalizations present in some of the preparations of the control group, which give the appearance of having a small lumen, could carry any significant amount of blood into the heart.

A striking contrast was noted between the carotids of the test animals where flow of blood into the right ventricle was effected and those ending blindly. This is shown in the illustrations. Fig. 5 is a comparison of the arteries proximal to the myocardial tunnel in a typical experiment. Intimal proliferation is insignificant in the test animal. The arteries within the tunnel and distal to it are compared in Figs. 6 and 7. Furthermore, in some of the test preparations there were strikingly prominent vessels about the implant as long as 6 months postoperatively, the presence and size of which could not be assigned to granulation tissue. These vessels which had been injected through the carotid implant were connected with the coronary circulation (Fig. 4). Further investigation of the factors involved in this encouraging finding are now in progress.

DISCUSSION

The discrepancies which have arisen in the several laboratories which investigated the procedure of arterial implantation into the myocardium are difficult to explain. Vineberg[1] has emphasized that two principles are of utmost importance in the maintenance of patency and the development of anastomoses. These are (1) the proper technique and (2) the presence of "need" or arterial insufficiency. With attention to these two features, he increased the number of his successful takes from 46 per cent to 75 per cent. Concerning the matter of technique, his descriptions are adequately detailed and lucid. It seems likely that those investigators assessing the operation were able to prepare their animals with satisfactory technical accomplishment. Why they were unable to find patent vessels remains a mystery. It may be noted, however, that those who were able to obtain persistently patent lumens in the implanted vessels followed an operative technique which varied somewhat from that described by Vineberg. Fuquay and associates implanted the left subclavian artery and left its 4 branches open in the myocardial tunnel. Sabi [illegible] nd [illegible] iates[1-]

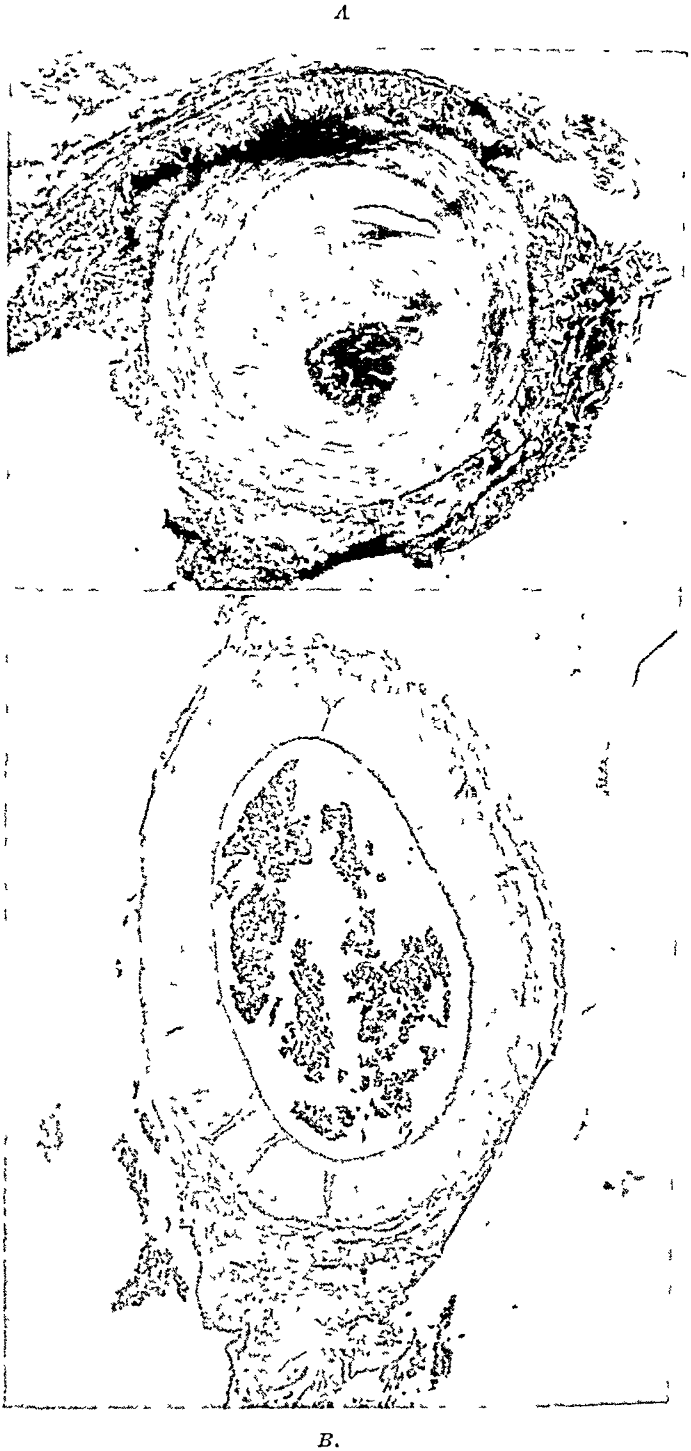

Fig 7 —Artery distal to tunnel in control animal showing obliteration of the lumen and organization of the thrombus after 7 weeks *B*, Wide open vessel distal to tunnel, proximal to right ventricular implantation, usual experimental result after 7 months

SUMMARY

1. The implantation of the left carotid artery in the myocardium of dogs by the standard technique resulted in intimal proliferation and obliteration of the lumen in 89 per cent of the animals by the end of one month.

2. By modifying the procedure so that the distal open end of the implanted artery is placed in the right ventricular cavity, the lumens of 100 per cent of the implanted vessels remained patent, some for as long as a 10-month observation period.

3. The trauma to the implanted artery which results from the repeated myocardial contraction was not responsible for the intimal proliferation.

4. Evidence is presented to indicate that the continuation of blood flow through the vessel is the most important factor in the maintenance of patency. The amount of flow necessary is still not known.

5. The chances for th[illegible] [illegible]t of anastomoses between the implanted artery and the coro[illegible] [illegible]ars to be improved in the ischemic heart muscle. Howeve[illegible] of the presence of anastomotic connections does not indic[illegible] amount of blood is being delivered to the myocardium.

Acknowled[illegible] [illegible]e is due Drs. John Clift, Bunket Lourvanij, Jerel Katz, and Mr [illegible]

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made a long tunnel into which they implanted an artery with multiple tangential openings. Smith and associates[14] used a nylon tube to convey the blood from the aorta through a ventricular tunnel, and they likewise had multiple perforations of the implanted portion of the tube. Carey and co-workers[4] anastomosed the end of the implanted artery to the left atrium. The results obtained by these investigators were similar to ours in showing that the majority of the implanted vessels do remain patent. All of these modifications appear to have one thing in common; namely, they are constructed so as to permit a continuous flow of blood through the implanted vessel. It is our opinion that this feature is the critical one in preventing the development of intimal proliferation and obliteration of the arterial lumen.

The second matter to which Vineberg directed attention is the receptive site. A myocardium with reduced arterial circulation, either as the result of surgical ligature or atherosclerosis, is presumably an area which will be more receptive to the blood furnished by an extracardiac vessel. On teleological grounds, therefore, the implanted artery will tend to remain patent and develop anastomoses with the existent arterial tree which is carrying a reduced volume of blood. In Vineberg's reported experiments, it may be noted in the normal hearts that the number of patent vessels was higher than the number with anastomotic connections (65 per cent as compared to 46 per cent) and in the ischemic hearts (76 per cent as compared to 71 per cent). While the latter differences are not significant, it is interesting to note that myocardial ischemia increased the number of patent vessels from 65 per cent in the control group to only 76 per cent. It would seem, therefore, that need is not too important a factor as regards the maintenance of patency. This argument is supported by the reports from other laboratories as well as our own findings that most of the arteries remained patent in hearts without myocardial ischemia.

The point which we wish to emphasize is that the majority of the implanted vessels will remain patent even in normal dog hearts as long as there is significant flow through the vessel. Presumably when anastomoses develop the flow has been adequate. However, the amount of blood flow required for the maintenance of patency is still unknown. Furthermore, the development of anastomotic connections with the coronary circulation does not necessarily indicate that a significant amount of blood is being supplied to the heart muscle. This point is substantiated by Vineberg's work[16, 17] showing a flow of only 6 or 7 c.c. per minute in 2 of 6 dogs despite the existence of anastomoses, the experiments of Sabiston and his co-workers[12] showing an average flow of only 5 c.c. per minute, and by our own observations that the presence of anastomotic connections is not always associated with the delivery of large volumes of blood to the heart. However, Vineberg may be correct in his opinion that implantation of an artery in ischemic heart muscle is more conducive to the development of anastomotic connections between the implanted vessel and the coronary circulation. The modification herein proposed, of implanting the open bleeding end of vessel into the right ventricle, fulfills the need for maintaining flow through the artery while anastomoses are being formed within the myocardium.

THE SURGICAL TREATMENT OF PECTUS EXCAVATUM

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THERE is general acceptance of the rationale for the surgical correction of the abnormality generally referred to as pectus excavatum, particularly when the deformity is marked. There remains uncertainty and disagreement regarding the indications for surgery when the deformity is mild or when the patient's physical development otherwise is unimpeded, and also in regard to the proper age at which surgery should be undertaken. As has been pointed out in a number of publications, the clinical manifestations and the indications for operative correction fall into three categories: cosmetic, functional, and orthopedic or postural, the last category being a function, in part, of the first two.

A number of variations in operative technique have been described since the important publication by Brown,[1] in 1939. These include the two procedures termed "simple" and "radical." The "radical" operation, which consists of the division or resection of the deformed costal cartilages, the removal or the division of the attachments of the xyphoid, and osteotomy of the anterior cortex of the body of the sternum with elevation of the gladiolus, has been modified in many ways since Brown described his operation. Many authors have emphasized the need for fixation of the elevated sternum to an external splint or prosthesis for variable periods of time.[1-5] Others have used methods of internal fixation which employ grafts of rib,[6] or cartilage,[7] or the use of wires[8] and Steinmann pins.[9] Ravitch,[10] in 1949, advocated a procedure in which no traction apparatus or internal splint is used to maintain the elevation of the sternum. He has since then employed such a procedure in a large number of cases with satisfactory results.[11]

Since 1948, we have operated upon 35 patients with funnel breast deformity. Five patients were adults. The youngest was 6 months of age, the oldest 42 years. External fixation by means of wire sutures, as was described by Brown, was utilized in only the first 2. No splinting, either external or internal, has been used in the remainder.

The purpose of this paper is twofold: to add emphasis to Ravitch's contention that external fixation of the mobilized sternum, by means of metal sutures or splints or by grafting, is not needed and to describe a modification of the operation described by Ravitch which has been performed upon 29 patients since 1954.

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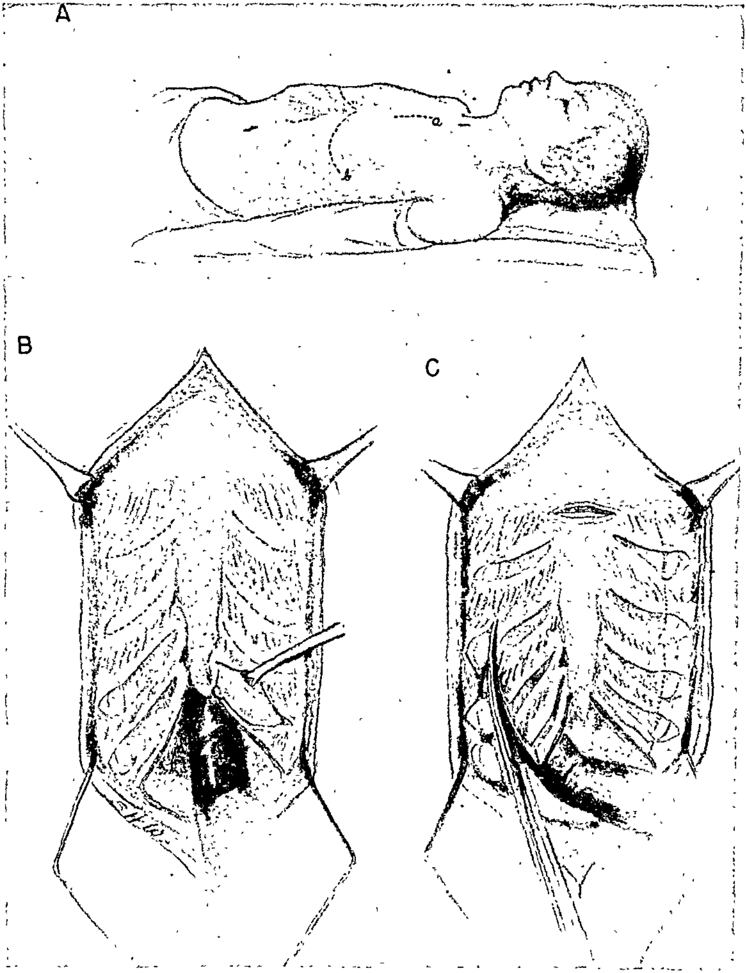

Fig. 1.—*A*, The incision may be in a vertical (*a*) or transverse (*b*) direction. Satisfactory exposure is obtained by either. We now prefer the transverse incision.

B, Excision of the costal cartilages is effected

C, The lines of incision through the entire che
anterior sternotomy.

TECHNIQUE OF OPERATION

The incision may be placed in a vertical direction, extending from the manubrium sterni to a point about midway between the xyphoid process and the umbilicus (Fig. 1, *A-a*), or it may be placed in a transverse manner, in which case it is extended across the xyphoid and to each anterior axillary line (Fig. 1, *A-b*). In the majority of cases we now prefer the transverse incision. The resulting scar of the midline incision may be unsightly and is usually high enough to be exposed through an open collar or low-necked dress.

The skin, subcutaneous tissues, and pectoral muscles are dissected away from the body of the sternum and the costal cartilages to such an extent that the greater portion of the anterior chest wall and the upper parts of the rectus abdominis muscles are exposed. This dissection should be extensive in order that all of the deformed portions of the costal cartilages are exposed. The cartilages are then excised subperichondrially (Fig. 1, *B*). It is important that this include all the deformed cartilages, whether or not the depression is symmetrical. In most cases the resection will include portions or all of the seventh cartilages and progressively shorter segments of the sixth, fifth, fourth, and third.

Following removal of the cartilages, the rectus muscles and the linea alba are freed from the xyphoid and from the perichondrium of the seventh cartilages. The xyphoid is not excised. The anterior cortex of the sternum is then transected at the level at which the posterior angle or curve begins; usually this is at the level of the third intercostal space and a wedge-shaped segment of the bone is excised to permit the sternum to be brought forward.

The sternum is elevated manually and the pleura is freed from the sternal borders and the endothoracic fascia by blunt dissection and is displaced laterally on each side. The parietal pleura on the right side is usually incised purposely to permit drainage into the pleural cavity postoperatively but the pleura on the left side is also often opened inadvertently. Incisions are then extended from the costal margins upward through the entire thickness of the soft tissue flaps, created by the subperichondrial resection of the costal cartilages, in a slightly curving line which is adjacent to the cut ends of the remaining costal cartilages (Fig. 1, *C*). The resulting soft tissue flaps, which have been left attached to the lateral borders of the sternum, are composed of endothoracic fascia, the intercostal muscle bundles, and perichondrium. Care has been taken to preserve the integrity of the internal mammary vessels superiorly, although they are often divided inferiorly as the rectus abdominis muscles are separated from the xyphoid and costal margins.

The soft tissue flaps are then brought outside the rib cage to overlap the costal cartilages and ribs laterally. They are sutured to the fascia overlying the intercostal muscles and to the perichondrium on each side, under moderate tension, with many interrupted sutures of silk (Fig. 2). These flaps, sutured as they are under moderate tension, act as taut "slings" to support the body

DISCUSSION

The results in the patients in whom this procedure has been carried out have been satisfactory on the whole. In the majority the sternum has been maintained in good position. In a few there has been slight retraction of the lower end of the sternum and, in these few patients, this has been noted not during the early postoperative period but always after a period of at least 6 weeks Thus, the use of an external prosthesis to maintain or "over-correct" the position of the sternum, if removed within the first 2 or 3 weeks after operation, would not have accomplished its purpose.

In a 4-year-old child, on whom the operation employed by Ravitch was carried out, there occurred gradual postoperative rotation of the sternum in such a manner that the right sternal border came to rest in a forward position beneath the skin as a visible and somewhat unsightly ridge or keel. This has not occurred following the technique described herein.

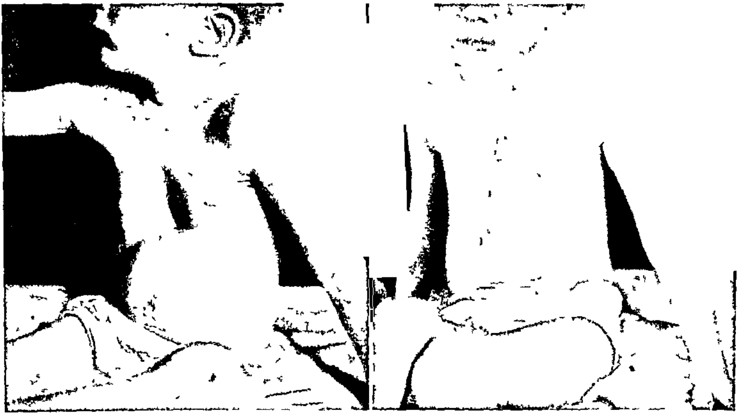
A. B

Fig 3—Appearance of anterior chest wall before operation (A) and 3 weeks following operation (B) Very slight retraction of the lower end of the sternum was noted several weeks later

The deformities in which this operative procedure has been employed have varied Usually the curve of the sternal body is concave on its mediastinal surface, while the xyphoid is turned forward at a sharp angle at its junction with the body Two children, one male and the other female, operated upon by us exhibited a "double curve" of the gladiolus, the anterior being convex as is usual at about the level of the third costal cartilage and slightly concave near its inferior end. Osteotomy was, therefore, performed at the two levels in order to flatten the sternal body and its position has been satisfactorily maintained in both cases. Lester[12] has recently classified deformities of the anterior chest wall and he calls attention to the fact that the position of the deformed of the sternum in a normal position. Interrupted sutures of silk are then placed to oppose the cut edges of the fascia and periosteum overlying the sternal osteotomy. No sutures are placed in the bone itself.

The cartilaginous xyphoid, which is sharply angulated forward, is partially divided transversely. It can then be easily molded or fractured so that it lies in a plane with the body of the sternum. The rectus abdominis fascia and linea alba are sutured to the xyphoid and to the lowermost margin of the lateral soft tissue flaps to complete the operation.

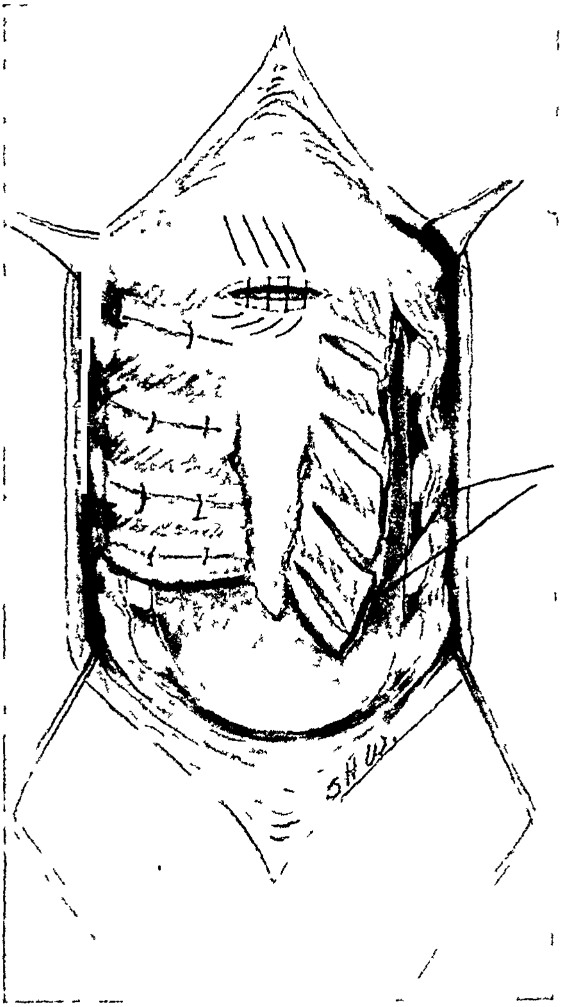

Fig 2.—The soft tissues, composed of perichondrium, intercostal muscles, and endothoracic fascia, are sutured under slight tension to the chest wall laterally

If the left pleural cavity has been opened, air is evacuated by means of a small catheter which is removed as the wound closure is completed. An inlying intercostal catheter is left in the right pleural cavity, through a stab wound in the lateral chest wall, for drainage of the mediastinum through the right pleural cavity. The pectoral fascia, subcutaneous tissues, and skin are approximated with fine silk sutures.

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costal cartilages and ribs is quite variable and often asymmetrical. He also notes that occasionally pectus excavatum will be associated with a posterior deformity of the upper end of the sternum and also, probably more frequently, with a flaring of the lower costal cartilages and ribs anteriorly. The unsightly flaring of the lower cartilages can often be improved or corrected entirely by the complete excision of not only the depressed medial ends of the lower cartilages but the flared lateral portions as well.

The operative procedure herein described has been carried out effectively and with considerable improvement, from a cosmetic standpoint, in one patient, a 16-year-old boy, with a marked protrusion deformity (pectus carinatum) of the sternum and anterior chest wall. Pulmonary function studies, carried out prior to and some weeks following the operation, revealed no change.

Pulmonary function has been measured in a number of children with pectus excavatum before and after operation. A number of these are still undergoing study. The results of these tests will be published later, but it does appear now that a definite increase in the pulmonary function, as related to the growth of the children, is measurable during periods of time which vary from a few weeks to several months following surgical correction of the deformity.

The value of the operative procedure from a functional standpoint has been well demonstrated in 3 children with cerebral palsy. Four such children, all with marked sternal depression and inspiratory chest wall retraction, have been subjected to operation. One died following cardiac arrest during operation. The remainder have been almost completely relieved from frequently recurring severe respiratory infections which had added much to the difficulty of their care prior to the corrective operations.

There have been no other deaths following operation except for the one recorded above. Complications have been few. One child, a 7-year-old girl, with cerebral palsy, suffered two prolonged generalized convulsive seizures on the first postoperative day. Her subsequent recovery was uncomplicated. Aspiration of small amounts of serous fluid from the wounds was necessary in 3 patients. There were no wound infections. The average time of hospitalization after operation has been 7 days.

SUMMARY

An operation for the correction of developmental deformities of the anterior chest wall is described in detail. The procedure involves an extensive dissection and mobilization of the sternum and the adjacent anterior chest wall and no external prosthesis or splint is employed. The postoperative care is simple. Complications have been few and the total period of hospitalization rather short. The procedure has been carried out on 28 patients with varied types of funnel chest deformity and on one patient with pectus carinatum or pigeon-breast. The results, from both the functional and cosmetic standpoint, have been satisfactory.

lymph sacs, which lie in the cervical region, grow downward along the trachea to the lungs. The left grows faster than the right and joins with it at the tracheal bifurcation, sending a large contribution to the right lung as well as to the left. From the retroperitoneal lymph sac, which lies in the upper part of the abdomen, arises the lymphatic supply of the lower third of the esophagus, the stomach, pancreas, spleen, liver, adrenals, and kidneys as well as the diaphragm and lower third of each lung. Usually 3 or 4 vessels, closely associated with those of the diaphragm, arise from the cephalic portion of the sac and grow dorsal to the stomach into the lung (Fig. 1). The vessels to the diaphragm supply that structure, and the pulmonary lymphatics extend into the pulmonary ligament about one third of the way to the hilum, dividing there into 2 branches

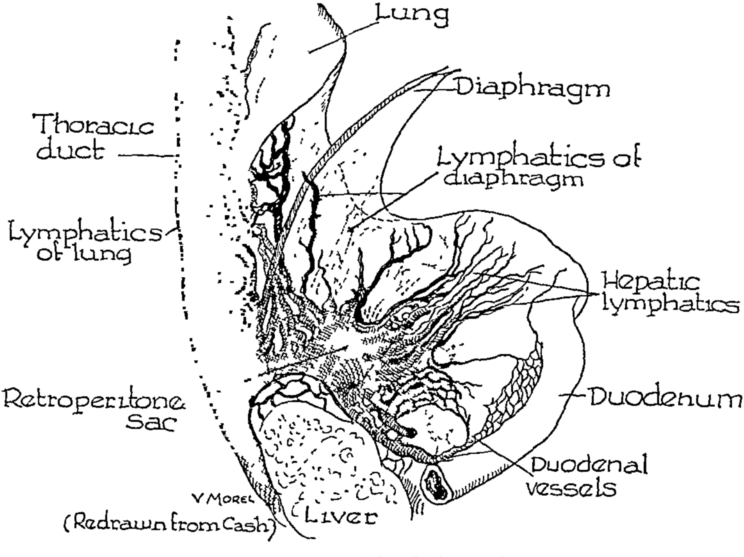

Fig 1.—Diagrammatic representation of lymphatic vessels arising from the retroperitoneal sac, from Cash [2] The lung and diaphragmatic lymphatic vessels originate in common and, as the latter spread over the diaphragm, the pulmonary lymphatics pass through it to reach the lower third of the lung Lymphatic vessels to the upper abdominal viscera also arise from this sac

One supplies the lateral and the other the diaphragmatic surface of the lower lobe. These grow into the lung and join with those extending out from the hilum

Since the vessels from the right and left lymph ducts persist in the adult as the right and left tracheobronchial chain, emptying into the respective jugulosubclavian trunks, it seems likely that the vessels from the retroperitoneal sac would also remain. If they do, and if flow through them to the abdomen is not impeded by valves, this mode of pulmonary lymph drainage would be

DIRECT LYMPHATIC CONNECTIONS FROM THE LOWER LOBES OF THE LUNG TO THE ABDOMEN

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INTRODUCTION

ABDOMINAL metastasis is relatively common in cancer of the lung. In the 3,047 collected cases of pulmonary cancer studied by Ochsner and DeBakey,[10] 78 per cent were metastatic to mediastinal lymph nodes, 33 per cent to the liver, 20 per cent to the adrenals, and 17 per cent to the kidneys. In 20 per cent of the 1,298 cases in which such information was recorded, metastasis to abdominal lymph nodes occurred. Although spread to abdominal lymph nodes is patently lymphatic, dissemination to abdominal organs may be by way of the lymph or blood vessels, by direct extension of the tumor, or by any combination of these. We still do not know which route of spread is most important and at what point, in the course of pulmonary carcinoma, metastasis occurs, despite the importance of these to therapy of the disease.[6]

Metastasis to abdominal nodes is usually considered to result solely from anomalous drainage caused by neoplastic invasion or adhesions of the lung to parietal or diaphragmatic pleura. However, Rouviere[11] described 2 specimens, and Mouchet and Nouriddine[9] one, in which the lymphatic vessels coursed from the lower lobes to a retroesophageal diaphragmatic node whose efferent ended in an abdominal para-aortic node. De Sousa,[5] in 2 specimens, found vessels from the left lung that ran without interruption to nodes of the gastric cardia or abdominal para-aortic nodes. In one of these, a vessel, which left the mediastinal surface of the right lower lobe, passed through 2 paraesophageal nodes before ending in an abdominal para-aortic node. This mode of lymph drainage could explain some of the cases of metastasis from pulmonary cancer to abdominal lymph nodes, but none of these authors seemed to consider it as other than an uncommon variant, and the existence of such a vessel is, in fact, specifically denied in a recent textbook of anatomy.[8] For these reasons, the possibility of such spread is seldom considered in the planning of therapy for patients with cancer of the lung.

The failure of more frequent demonstration of such routes of intra-abdominal pulmonary lymphatic drainage is surprising in view of the embryologic development of pulmonary lymphatic vessels. Cunningham[4] demonstrated that the pulmonary lymphatic vessels arise from 3 sources: the right and left jugular lymph sacs and the retroperitoneal lymph sac. The vessels from the jugular

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nodes and all specimens displayed drainage to this area through the usually described channels. Only those vessels that coursed to the abdomen will be described.

When present, these vessels arose from the diaphragmatic surface or the lower medial surface of the lower lobe. Within the pulmonary ligament, they invariably coursed near its lower free edge, following the ligament to its attachment to the diaphragm. Usually the several small vessels that left the lung united into a single trunk. This continued as a single trunk or it divided into two or more channels. These then passed to the abdomen without interruption, or joined the infrapericardial nodes or the posterior diaphragmatic lymphatics before terminating in the upper part of the abdomen.

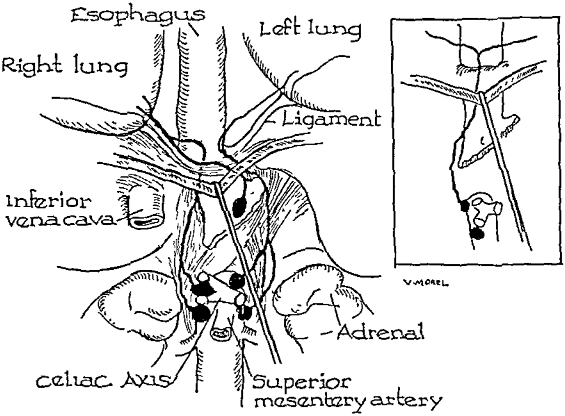

Fig. 2.—Diagrammatic representation of pulmonary lymphatic vessels that drain to the celiac nodes. These vessels usually pass through the esophageal hiatus, then run along either crus to the aortic nodes near the celiac axis. They may pass directly through the diaphragm. Occasionally they terminate in or send a branch to a node of the cardia. The inset shows the union of vessels from the right and left lungs observed in one specimen.

In Fig. 2 is shown the general path of those vessels that coursed uninterruptedly to abdominal nodes. These were observed issuing from the right lower lobe in 2 of the 15 successful injections of this lobe and from the left lower lobe in 3 of 12 successful injections. The vessels leaving either lobe passed in the pulmonary ligament to reach the esophagus slightly above the hiatus or ran on the diaphragm for a short distance before traversing the hiatus anterior, posterior or lateral to the esophagus. In one specimen the vessel from the left lower lobe passed through the diaphragmatic musculature instead of the hiatus. Within the abdominal cavity the vessels left the esophagus to run down either crus to the nodes about the celiac axis. Some terminated in, or sent a branch to, the nodes of the cardia before ending in the celiac nodes.

The inset in Fig. 2 shows the union of the vessels from the right and left lower lobes which was observed in one specimen. These vessels joined anterior

Cunningham[4] suggested, have considerable clinical significance. Although previous studies indicate only rare persistence of this fetal state, the poorer prognosis of cancer of the lower lobe[1] (13 per cent of patients with cancer of the lower lobe survive 2 years as compared with 22 per cent of those with carcinoma of the upper lobe) and the extensive spread of pulmonary carcinoma to the upper part of the abdomen suggest that such vessels exist commonly. The purpose of the present study was to determine the status of these channels, the extent to which they persist, and their character.

MATERIALS AND METHODS

Twenty-three stillborns and neonates, which were anatomically normal, were obtained after storage in a morgue refrigerator for 72 hours. In some, injection and study were done immediately but most were preserved by freezing at –10° F. for several days to months. This freeze storage had no noticeable effect on the injection.

Since lymphatic vessels are more successfully injected when the tissues are warmed,[14] all specimens were thawed and immersed in warm water to bring them as closely as possible to body temperature. The ribs were then removed to obtain good exposure of the lungs but the sternum and costal arches were left in place to support the mediastinum and diaphragm in correct anatomic relationships. After the lungs had been inflated through a tracheostomy, India ink, diluted with 3 parts of distilled water, was injected subpleurally through a 26-gauge needle, according to the technique of Simer.[14] The lower third of the respective lower lobe, the region that receives the vessels from the retroperitoneal sac in the embryo, was injected carefully with particular attention to the diaphragmatic and medial surfaces near the pulmonary ligament. This area is difficult to inject because the pulmonary ligament must not be stretched or twisted during the injection. If it is, the vessels that course in its lower free edge may be blocked and thus not visualized; these vessels course to the abdomen.

In successful injections, the pleural lymphatic network was filled in a short time and the ink flowed rapidly within the pleural collecting vessels. The lung was then gently stroked to aid the flow of ink along the vessels. Usually only 1 c.c. of ink was injected as this gave adequate filling of lymphatics and larger amounts disrupted the substance of the lung. The ink-filled vessels that left the lung were traced with blunt and sharp dissection, loupe binoculars or a dissecting microscope being used. Whenever possible, the node into which the vessel drained was also injected. However, the small size of these nodes made this most difficult and was usually unsuccessful. The specimens were then preserved in 10 per cent formalin for further study; sketches of the course of the ink-filled vessels were made.

OBSERVATIONS

The lymphatic vessels in the right lower lobe were demonstrated in 15 specimens and those in the left in 12 specimens. The criterion of successful injection was the filling of lymph vessels that emptied into tracheobronchial

The general course of the vessels that ran on the posterior diaphragm is shown in Fig 4 These vessels were demonstrated in 4 specimens, all from the right lower lobe As they coursed posteriorly or posterolaterally, their branches joined with those of the intrinsic diaphragmatic lymphatics

DISCUSSION

Direct lymphatic communication between the lung and the upper abdominal viscera and lymph nodes is either by way of vessels that pass through the esophageal hiatus to the celiac nodes or by confluence of lymph vessels from the lung with those of the posterior diaphragm Although the vessels that pass through the esophageal hiatus may have no direct communication with abdominal organs other than the stomach, those blending with the posterior diaphragm are, through its lymphatics, in direct continuity with the vessels of the liver, adrenal gland, and kidney before draining ultimately to the celiac and para aortic lymph nodes

From both lower lobes, the subpleural collecting vessels that course directly into the abdomen arise from or just above the diaphragmatic surface of the lung and run in the pulmonary ligament near its lower free edge Traveling medially, they reach the esophagus slightly above the hiatus Then paralleling the esophagus, they traverse the hiatus anterior, posterior, or lateral to the esophagus, either separately or as a single trunk resulting from the union of the lymphatics from each lung Within the abdominal cavity the vessels leave the esophagus to run down the abdominal surface of either crus, frequently skirting the adrenals, and end in the nodes about the celiac axis and aorta

The diaphragmatic nodes frequently interposed in the course of the vessel from the right lung, and occasionally from the left, lie near the esophageal hiatus, to the left of the inferior vena cava These nodes, the infrapericardial nodes, which were described and named by Sledziewski,[15] vary in number (from 1 to 5) and size but always lie sandwiched between the pericardium and the diaphragm and thus are not readily visualized As did Sledziewski, I found multiple efferents from these nodes Some ascend to tracheobronchial nodes, others pass backward to retroesophageal nodes from which they may empty into the thoracic duct or to intra abdominal para aortic nodes[9] [11] Others pass through the esophageal hiatus or diaphragm to end in nodes of the cardia or celiac axis and so carry pulmonary lymph to the abdomen

Of great importance as a route of metastasis of pulmonary tumor to abdominal viscera are those pulmonary lymphatic vessels that blend with the intrinsic lymphatic vessels of the diaphragm These vessels, which reach the diaphragm through the pulmonary ligaments, and then pass posteriorly or posterolaterally on the diaphragm, were found only from the right lung, this confirms the observations of Sappey[13] and Cruikshank[3] Their importance is that the lymphatic network of the posterior diaphragm joins not only both diaphragmatic surfaces but some of the lymphatics draining the adjacent viscera—lungs, liver, adrenals, and kidneys—into a common interconnecting pool, which drains eventually to nodes about the upper end of the abdominal aorta[12] to the esophagus and above the diaphragm. A single trunk coursed through the hiatus to the right of the esophagus and then ran down the abdominal surface of the right crus to end in 3 nodes about the celiac axis.

Vessels that joined the diaphragmatic nodes were observed in 6 specimens of the right lower lobe and in one of the left lower lobe. These nodes and their connections are illustrated in Fig. 3. Because these nodes are often so small, it was not always possible to trace their efferent connections. However, those demonstrated ascended to tracheobronchial nodes or coursed backward to a retroesophageal node or passed through the diaphragm to nodes about the celiac axis.

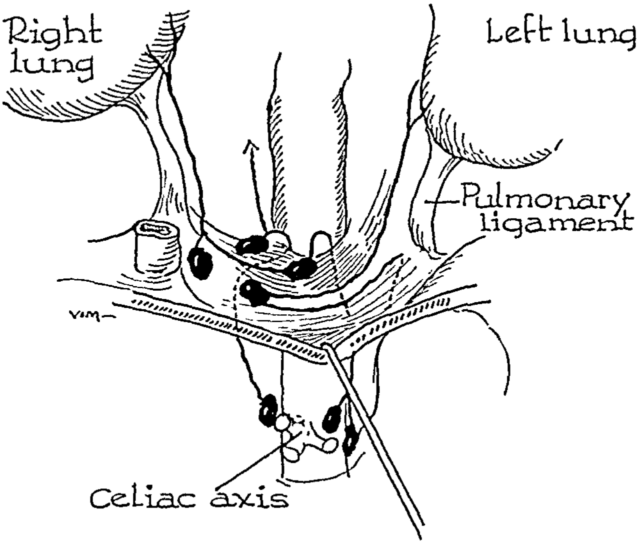

Fig. 3.—Diagrammatic representation of the infrapericardiac nodes and their connections. These lie between the pericardium and diaphragm and receive lymph from the liver as well as the lungs. Their efferents are to abdominal peri-aortic nodes, nodes of the cardia and carinal nodes. Lymph from the right lung drains to these nodes more often than does that of the left.

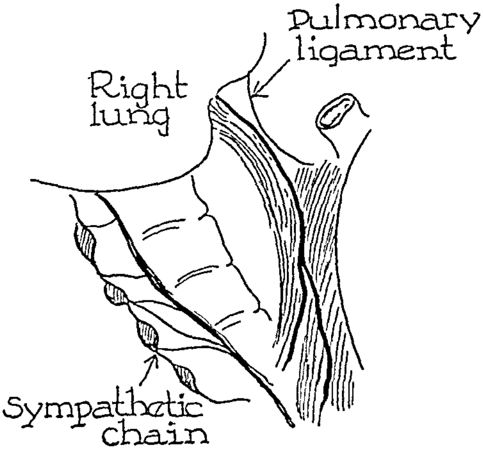

Fig. 4.—Diagrammatic representation of the pulmonary lymphatic vessels that join the posterior diaphragmatic lymphatic vessels. These were observed from the right lung only. The diaphragmatic vessels, in turn, join those of the liver and adrenal before emptying into the para-aortic and celiac nodes.

Vessels from the right lower lobe that coursed to the celiac nodes without interruption were observed in 2 specimens They entered the abdomen by passing through the esophageal hiatus and then ran on either crus to the nodes about the celiac axis Other lymphatic vessels from the right lower lobe, which ran in the pulmonary ligament to the diaphragm, were demonstrated in 9 specimens On reaching the diaphragm they either joined with the lymphatic vessels of the posterior diaphragm or ended in a pre esophageal diaphragmatic node Some of the efferents of this node group ended in nodes about the celiac axis

From the left lower lobe, vessels that coursed to the abdomen were observed in 3 specimens They ran medially in the pulmonary ligament to the esophagus, entered the abdomen by traversing the esophageal hiatus and ran on either crus to nodes about the celiac axis

The communications of the lymph vessels of the posterior diaphragm with those of the lungs, liver, adrenals, kidneys, and abdominal lymph nodes have been briefly reviewed

From these observations the conclusion is drawn that pulmonary lymph normally drains to the abdomen and that early, direct metastasis of pulmonary cancer to the abdomen, through the vessels demonstrated, may account for the frequency of intra abdominal metastases in pulmonary cancer

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In describing the lymphatic drainage of the diaphragm, Rouviere[12] emphasized that its extensive subpleural and subperitoneal lymph networks are intimately connected by penetrating vessels, which drain to the subpleural collecting trunks. In the posterior half of the diaphragm, these pass through the diaphragm to end in nodes about the cardia, the upper part of the abdominal aorta and the origin of the inferior phrenic arteries. These nodes are the ones into which the uninterrupted channels from the lung empty.

In addition, Rouviere stressed that some of the lymphatic vessels of the liver and adipose capsule of the kidney and adrenal ascend to penetrate the diaphragm and join the subpleural collecting trunks while others join the collecting trunks of the diaphragm which course along the inferior phrenic artery. The continuity of these collecting trunks with the adrenal lymphatics is well illustrated in Higgins and Mills'[7] physiologic study of absorption from the peritoneal cavity.

The present study has demonstrated that the diaphragm is not a barrier to, but rather a junctional link between, the lymphatic vessels draining the thoracic and upper abdominal viscera and that, in the absence of adhesions between parietal and diaphragmatic pleura, there are routes through which pulmonary lymph drains to abdominal nodes.

The role of this functional continuity in the metastatic pattern of cancer of the lung is readily apparent. Since the celiac and para-aortic lymph nodes are the first echelon of nodes of pulmonary lymph drainage, a tumor of the lung can spread to the abdominal lymph nodes as readily as to mediastinal nodes—and this spread may not be evident at thoracotomy. Through the blending of lymph from the lungs, liver, adrenals, and kidneys in the posterior diaphragm, neoplastic cells within the diaphragmatic lymphatic vessels can spread directly to these organs. Metastatic tumor from the lungs, growing in celiac or para-aortic lymph nodes, can pass within the afferent vessels of these nodes to any of the upper abdominal organs (the reverse may also occur). Lymph-borne abdominal metastasis in cancer of the lung may occur early and must be suspected in every patient with cancer of the lower lobes of the lung.

SUMMARY

The lymphatic drainage of the lower lobes of the lung was studied to determine if any lymphatic vessels from the lung course directly to the upper part of the abdomen. Twenty-three stillborns and neonates were preserved by refrigeration until used, when they were warmed and the lungs were inflated. India ink, diluted 1:3 with distilled water, was injected subpleurally into the mediastinal and diaphragmatic surfaces of the lobes and the ink-filled vessels were traced to their terminations. The anatomic relationships of the pulmonary ligament were carefully maintained during injection.

Eighteen specimens were successfully injected. The lymphatic vessels of the right lower lobe were demonstrated in 15 of these and of the left lower lobe in 12.

distress In the third interspace, about 5 cm from the midline, there were two small lacerations A small amount of subcutaneous emphysema was present over the left upper chest A harsh, Grade 4 to 5, continuous murmur was heard throughout the entire precordial area but most loudly in the second left anterior interspace, approximately 1 to 2 cm to the left of the midsternal line A continuous thrill was palpated in the same area The hemoglobin determination and erythrocyte count were normal and there was a slight leukocytosis Urinalysis showed a reducing substance to be present in the urine, however, a glucose tolerance test done later was within normal limits Cardiac fluoroscopy revealed a slight increase in the size of the pulmonary conus and a questionable enlargement of the left ventricle The pulmonary arteries were prominent, but the peripheral lung fields were fairly clear An irregular, opaque foreign body, measuring 1 cm in greatest diameter, was present in the left anterior mediastinum (Fig 1) On lateral view, the foreign body was seen just beneath the sternum

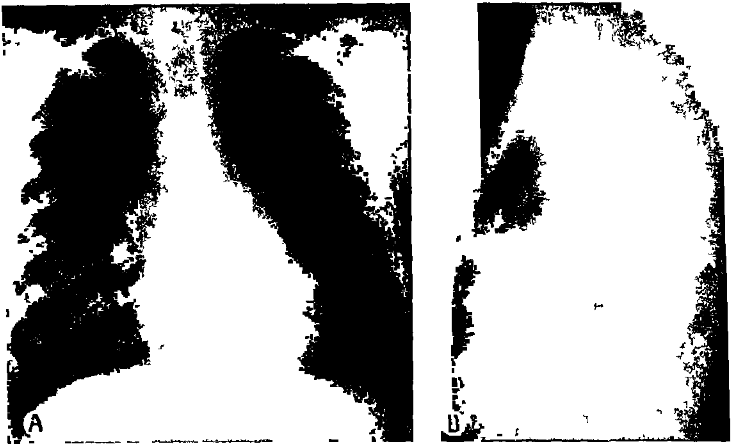

Fig 1—Roentgenograms taken at the first admission

During the 4 days after admission, the patient's rapid pulse rate gradually slowed to an average of 85 per minute The blood pressure stabilized at about 100/50 to 100/60 mm Hg The low grade fever present on admission also subsided The patient's dyspnea disappeared Our initial impression was that the continuous murmur resulted from the injury to the chest Later, however, we arrived at the erroneous conclusion that the patient more likely had a congenital patent ductus arteriosus, and that the injury was unrelated The site of injury healed and the patient was discharged

He was readmitted on December 4 The blood pressure was 95/50 mm Hg and the pulse was 92 per minute The same cardiac findings were present On consultation the pediatric cardiologist still felt that the patient had a typical patent ductus arteriosus Therefore, on December 6, the patient's left thorax was explored A patent ductus was not present The continuous thrill could be palpated through the pericardium anteriorly in the region of the base of the pulmonary artery or right ventricular outflow tract In this area there was dense scar tissue and adhesions of the pericardium to the anterior chest wall Since it seemed likely that open cardiotomy with bypass of the heart and lungs would be necessary to cure the lesion, the pericardium was not opened at this time and the chest was closed The patient's postoperative course was good and he was discharged from the hospital

SURGICAL REPAIR OF A TRAUMATIC AORTIC-RIGHT VENTRICULAR FISTULA

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AORTIC-RIGHT ventricular fistula is a rare cardiac lesion. Sawyers and his associates[1] recently reviewed the literature since 1840, and found only 19 cases. Since that report, a few instances in which surgical closure was attempted or carried out have been reported.[2, 3, 4]

Aortic-right ventricular fistulas usually occur following rupture of an aneurysm of the right coronary sinus into the ventricular chamber. Aneurysms of the sinus of Valsalva are most frequently of congenital origin, although some have occurred following syphilitic or bacterial infection. In the absence of endarteritis, no symptoms occur until the aneurysm ruptures. At that time, there is usually chest pain and difficulty in breathing.[5, 6] Often, a whirring sensation is felt in the precordial region. Over the next few days the symptoms generally decrease in their intensity. The patient then enters a temporary, relatively symptomless, phase. The average life expectancy following development of a fistula, however, is usually about one year. Death most frequently occurs following congestive heart failure. In a few cases, bacterial endocarditis develops and death ensues from an overwhelming bacteremia. In the average case, death occurs in the third decade of life.

Trauma must be a most unusual cause of aortic-right ventricular fistula. Interventricular septal defects and aortic-pulmonary fistulas have occurred infrequently following cardiac trauma. Gerbode[7] reported a case of traumatic aortico-atrial fistula caused by a bullet. We have been unable to find the record of any case of traumatic aortic-right ventricular fistula.

This report is concerned with an aortic-right ventricular fistula which occurred following a gunshot wound of the chest. The eventual clinical diagnosis was confirmed by cardiac catheterization, cineradiography, and by exploration of the right ventricle at the time of surgical repair.

CASE REPORT

The patient was a 15-year-old white boy who was first admitted to the Indiana University Medical Center on Sept. 23, 1956. On the morning of admission, the patient was hammering a blank 30-30 cartridge when it exploded and struck him in the chest. He was severely dyspneic on arrival at his local hospital. A loud cardiac murmur was heard. He was transferred to the Medical Center 10 hours after injury. At that time, his pulse was 140 and regular, respirations 22, and temperature 100.8°. The blood pressure was recorded as 120/0 mm. Hg on one occasion and 120/30 on another. After the first 4 hours of hospitalization, the diastolic pressure gradually increased and the blood pressure ranged from 110/40 to 118/60 mm. Hg. The pulse remained at 140 to 150 per minute. He was in acute

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the coronary arteries After cardiac standstill was achieved, the right ventricle was opened through a vertical incision made downward from the base of the pulmonary artery The fistula was easily seen (Fig 2) It measured about one centimeter in diameter A narrow fibrous band lined the edge of the defect A Kelly clamp was easily passed through this opening and emerged within the aorta in the region of the right coronary sinus of Valsalva The defect was closed with a running suture of 5 0 silk and reinforced with 2 interrupted 5 0 silk sutures The right ventriculotomy was then closed After the clamp was removed from the aorta, ventricular fibrillation began and four electric shocks were necessary to convert the heart action to a normal rhythm A blood pressure of 120/90 to 130/90 mm Hg was obtained almost immediately The pulse was 100 per minute After the cannulas were removed, a large part of the scarred anterior pericardium was excised

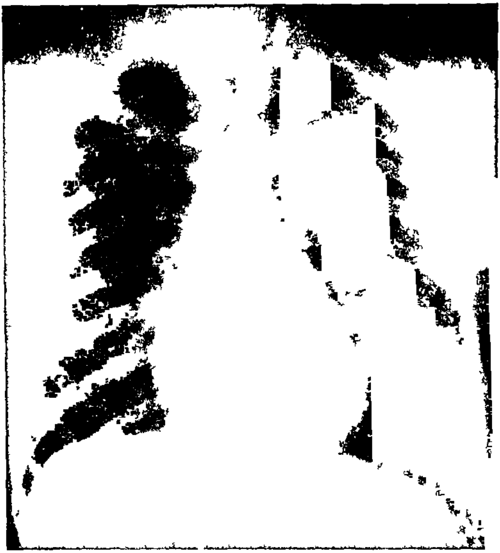

Fig 3 —Roentgenogram of chest taken a few weeks after operative repair

The patient's immediate postoperative course was good The blood pressure ranged from 130/80 to 120/80 mm Hg with a pulse rate of 120 per minute Two days after operation, he had a generalized convulsion involving all extremities and was mentally confused The serum sodium was found to be 123 mEq with a blood pH of 7 64 The electrolyte abnormalities were corrected over the next 2 days and, on March 17, he was coherent and rational He was discharged from the hospital 15 days after operation in good condition

The patient gained 13 pounds in the first 8 weeks after operation His exercise tolerance greatly increased Seven months after operation, the blood pressure varied from 118/70 to 105/68 mm Hg with a pulse rate around 85 The cardiac sounds were good and no thrill or murmurs were present A roentgenogram of the chest showed the heart to be normal in size and configuration (Fig 3) Fluoroscopy of the patient's body revealed a thin metallic fragment, measuring about 1 by 1 5 cm in size, located about 3 cm to the left of the first sacral vertebral body (Fig 4) This fragment undoubtedly created the cardiac fistula and then passed into the aortic stream as an embolus to lodge in the left common iliac, external iliac, or hypogastric artery The arterial pulsations in the l[illegible]le were excellent It is planned to remove the metallic fragment in the near future

While home, he did not do well. He had frequent attacks of pulmonary congestion. Some were thought to be upper respiratory infections but others were clearly due to cardiac failure. Because his physical condition seemed to be gradually deteriorating, he was readmitted on March 9, 1957. Physical examination was the same as before. The blood pressure varied between 114/50 and 110/60 mm. Hg. The pulse was 90 per minute. On cardiac catheterization, oxygen saturation of the blood increased from 79 in the atrium to 95 per cent in the region of the right ventricular outflow tract. The pulmonary artery and right ventricular pressures were not increased, both being around 26 mm. of mercury. Cineradiography demonstrated dilution of the opaque media in the outflow tract of the right ventricle and this presumably was due to a left-to-right shunt at this point. The fistulous tract itself was not seen. These data demonstrated a left-to-right shunt into the right ventricle. They did not permit a clear differentiation between a ventricular septal defect and an aortic-right ventricular fistula. Taking everything into consideration, we reached the conclusion that the best tentative diagnosis was a fistulous tract between the right sinus of Valsalva and the right ventricular outflow tract.

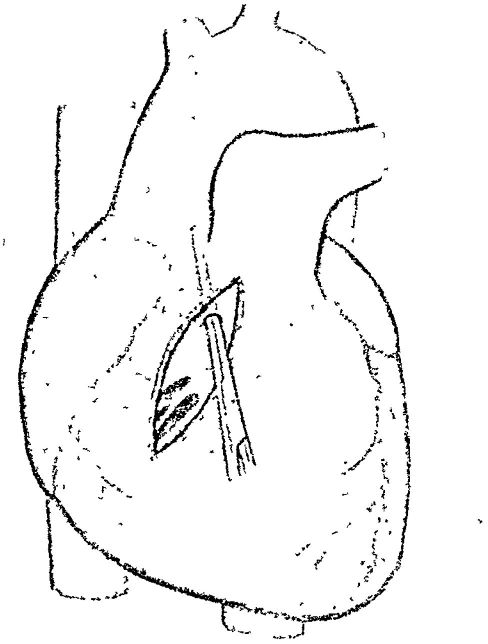

Fig. 2.—Drawing showing the fistulous tract as observed through the right ventriculotomy incision. A large clamp has been placed through the tract into the aorta. At operation, the cardiac landmarks were obscured by the overlying pericarditis.

On March 13, a bilateral anterior thoracotomy was carried out in the fourth interspace. The sternum was split transversely. Before operative repair, the blood pressure ranged from 130/60 to 140/70 mm. Hg. The pulse rate was 80 per minute. There were numerous adhesions between the anterior chest wall and the pericardium. The pericardium was adherent to the heart and great difficulty was encountered in dissecting it free. Even after this was accomplished, it was impossible to recognize superficial cardiac landmarks such as the coronary arteries. Only the general outline of the right ventricle could be defined. The point of maximum thrill was located about one inch below the base of the aorta over the right ventricular outflow tract. Here, recent scar tissue, about 0.5 cm. in diameter, protruded from the ventricular wall. The venae cavae and the left femoral artery were cannulated and cardio pulmonary bypass was begun at the rate of 35 c.c. per kilo per minute. Twenty-five per cent potassium citrate solution dissolved in blood in a ratio of 2 c.c. to 18 c.c. was injected into the base of the aorta after it was clamped. Over 100 c.c. of this solution were required to arrest the heart. In retrospect, a substantial amount of the potassium citrate probably went through the fistulous tract into the right ventricle instead of entering aneurysm of the sinus of Valsalva into the right heart. In most cases, if the patient survives the initial insult, there is a period of a few weeks to a few months during which he remains in relatively good health. It is during this time that surgical therapy can best be carried out. Later, the patient tends to become progressively more disabled and the risk of operation undoubtedly increases. If surgery is deferred until after bacterial endocarditis has become well established, the risk will be even greater.

The surgical technique for closure of these defects is not yet well established. Our patient, treated by suture closure of the right ventricular portion of the fistula, has done well. Others have reported success with the same technique. In dealing with a coronary sinus right atrial fistula, Baker, Hanson, and Morrow[8] plugged the fistulous tract with molded Ivalon sponge. They feel that this technique will more effectively prevent recurrence of the lesion by at least partially obliterating the aneurysm. Ventricular suture of the defect has given good results in our case and in several others. Although it would have been easy to have inserted a piece of Ivalon sponge into the fistulous tract in our patient, we decided against this maneuver since we could not definitely rule out involvement of the proximal portion of the right coronary artery and did not wish to risk occluding it.

SUMMARY

No case of aortic right ventricular fistula of traumatic origin has been found in the literature. A case report is presented in which a 15 year old boy sustained a gunshot wound of the chest resulting in penetration of the right ventricle by a metallic body which passed into the aorta, producing a fistulous communication between the two. All the signs of a patent ductus developed including those of cardiac failure. At operation, using a cardiac bypass, the right ventricle was incised and the opening closed with complete recovery.

We wish to express our appreciation to Dr. Paul Lurie who performed the catheterization studies and to Dr. Eleanor Clay who referred the patient for treatment. We are very grateful to Dr. Emile Holman who suggested the possibility of a second metallic foreign body which had caused the cardiac injury and passed into the distal arterial circulation.

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DISCUSSION

The aortic-right ventricular fistula, in this case, was secondary to the perforating wound of the left anterior chest. Until the metallic embolus was discovered by fluoroscopy and x-ray study, the mechanism of the injury was puzzling. It now seems clear that the fragment penetrated the right ventricular wall at the point where the superficial scar tissue was evident on exploration and then passed through the underlying ventricular septum directly into the aorta and from there distally into the iliac arterial bed. Whether it entered the aorta through the main right coronary artery or through the wall of the right coronary sinus of Valsalva remains a question, though in view of the relatively large size of the foreign body and of the fistulous tract the latter seems much more plausible. A clamp could be passed easily through the fistula into the aortic sinus. It was concluded that it probably connected the sinus and the ventricular chamber directly.

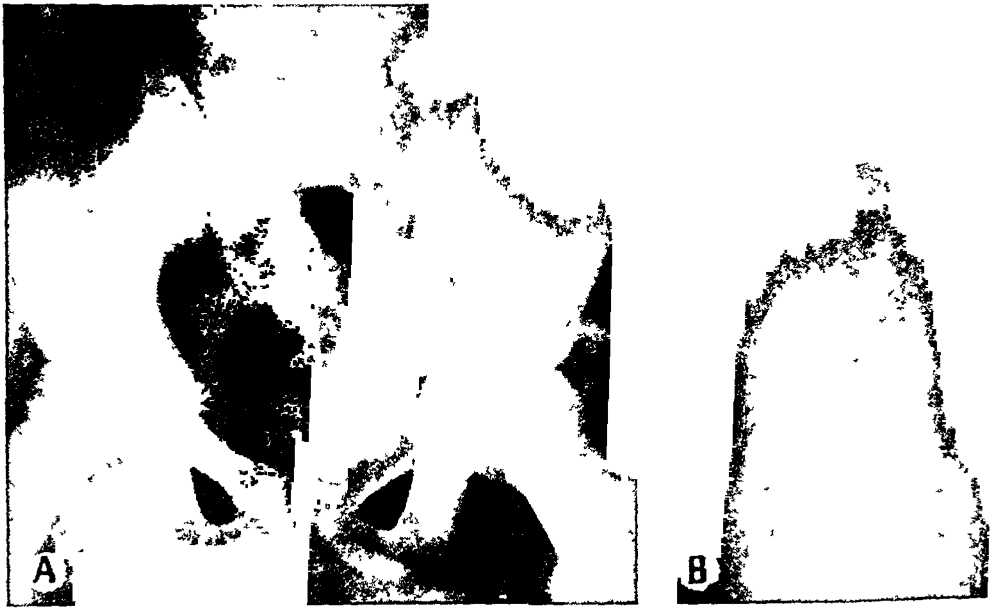

Fig. 4.—Roentgenograms of the pelvis show an embolic metallic fragment located to the left of the first sacral vertebra

The course of the patient's blood pressure is of interest. Shortly after injury the pulse pressure varied from 90 to 120 mm. Hg. This gradually decreased but remained greater than normal. During anesthesia and before operative repair, the pulse pressure was 70 mm. Hg. Immediately after operative repair, the diastolic pressure increased and the pulse pressure fell to from 30 to 40 mm. Hg. Since recovery from operation, pulse and blood pressure have been normal. Altogether his pulse and blood pressure changes are those which might be expected from any vascular fistula between a high and a relatively low pressure area.

Any aortic-right ventricular fistula, regardless of etiology, would probably produce the same clinical picture as that described following rupture of an with a pressure-measuring apparatus (Electromanometer, Elema) coupled to a direct-recording, 2-lead electrocardiogram apparatus (Mingograph 23, Elema). When the operative wound has been closed, the catheter is removed, generally about 6 hours after its introduction into the heart.

Postoperative examination, including cardiac catheterization, is carried out not earlier than one month after the operation.

The peripheral blood pressure is measured bloodlessly with a sphygmographic cuff coupled to a mechanical manometer.

The pulse rate is calculated from the ECG-curves.

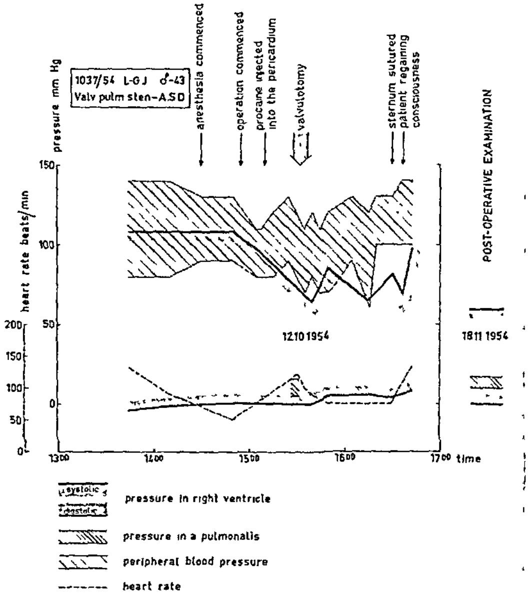

Fig. 1.

MATERIAL

The pressure measurements have been carried out in this way during the operation for 5 valvular pulmonary stenoses, 1 atypical patent ductus arteriosus, and 1 case of coarctation of the aorta. The recordings from 2 patients with pulmonary stenosis were technically unsatisfactory; the pressure values in the other cases are represented graphically in Figs. 1-5.

Fig. 1 shows the recordings of pressure changes in the right ventricle in connection with valvulotomy performed on an 11-year-old boy with valvular

INTRACARDIAC PRESSURE MEASUREMENT DURING OPERATION FOR CONGENITAL HEART DISEASE

Value of the Method for Assessing the Effect of the Operation

Åke Lundberg, M.D., Stockholm, Sweden

INTRODUCTION

During the year 1954 an increasing number of patients with valvular pulmonary stenosis without aortic overriding were operated upon at the surgical department of our hospital. The operation was performed transventricularly, according to Brock,[2] so that the valvulotomy took place without direct control of the eye. In these circumstances, the idea of being able, at least indirectly, to observe the result of the splitting of the valve in the course of the operation seemed attractive.[4]

With this end in view, attempts had been made in various quarters to get some idea of the reduction in pressure in the right ventricle in the course of operation. This was done with isolated pressure recordings, either via a puncture needle through the wall of the right ventricle, or with the help of a catheter introduced into the right ventricle through the operator's incision in the heart wall.

Pressure measurements via a catheter introduced into the heart before the operation seemed to me more advisable from several points of view.

1. The pressure values could then be obtained at any time—immediately before, during, or immediately after the operation, and not only during the phase in which the heart was directly accessible for the surgeon's manipulations.
2. Further damage to the ventricular wall through the puncture could thus be avoided.
3. The operator and his assistants need not be disturbed by interference in the pressure-measurement procedure.
4. It would be of great interest to obtain, during certain stages of the operation such as the valvulotomy, *continuous pressure recordings* from the right ventricle.

METHOD

Before the operation, while the patient is still conscious and has been given only the usual premedication (morphine scopolamine), a catheter (Cournand No. 7 or 8) is introduced via the cubital vein or vena saphena magna and disposed with the point in the desired position, as a rule the right ventricle. The child is then transferred to the operating theater, where the catheter is connected

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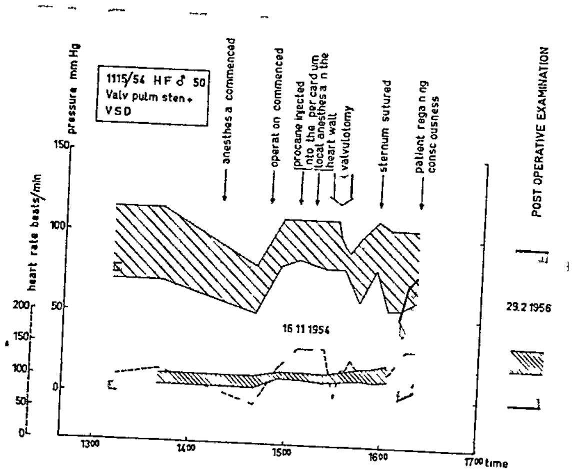
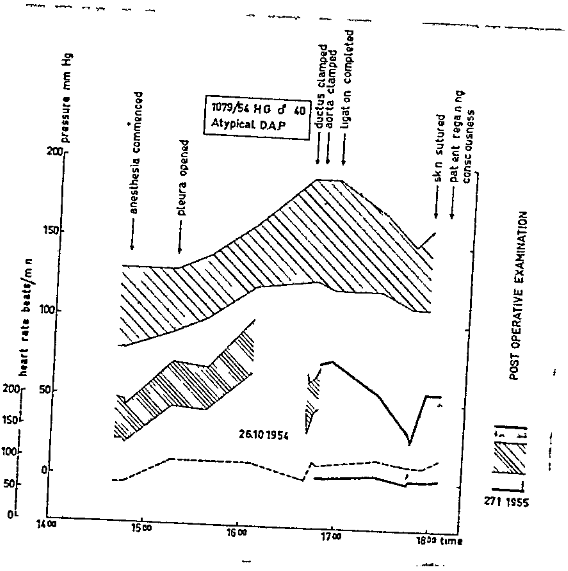

1115/54 HF ♂ 50
Valv pulm sten+
VSD
pressure mm Hg
heart rate beats/min
150
100
50
0
200
150
100
50
0
anesthesia commenced
operation commenced
procaine injected into the pericardium
local anesthesia in the heart wall
valvulotomy
sternum sutured
patient regaining consciousness
POST OPERATIVE EXAMINATION
16 11 1954
29.2 1956
13^00
14^00
15^00
16^00
17^00 time

Fig 3


1079/54 HG ♂ 40
Atypical DAP
pressure mm Hg
heart rate beats/min
200
150
100
50
0
200
150
100
50
0
anesthesia commenced
pleura opened
ductus clamped
aorta clamped
ligation completed
skin sutured
patient regaining consciousness
POST OPERATIVE EXAMINATION
26.10 1954
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14^00
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17^00
18^00 time

Fig 4

pulmonic stenosis combined with a slight defect in the auricular septum. Even before the start of the valvulotomy, the pressure sank from 110 to 80 mm. Hg after the inception of narcosis. During the splitting of the valve, the pressure dropped somewhat further, to show varying values as the patient began to regain consciousness.

The postoperative examination one month later shows, in the waking state, a pressure lower than the lowest pressure measured in the course of the operation.

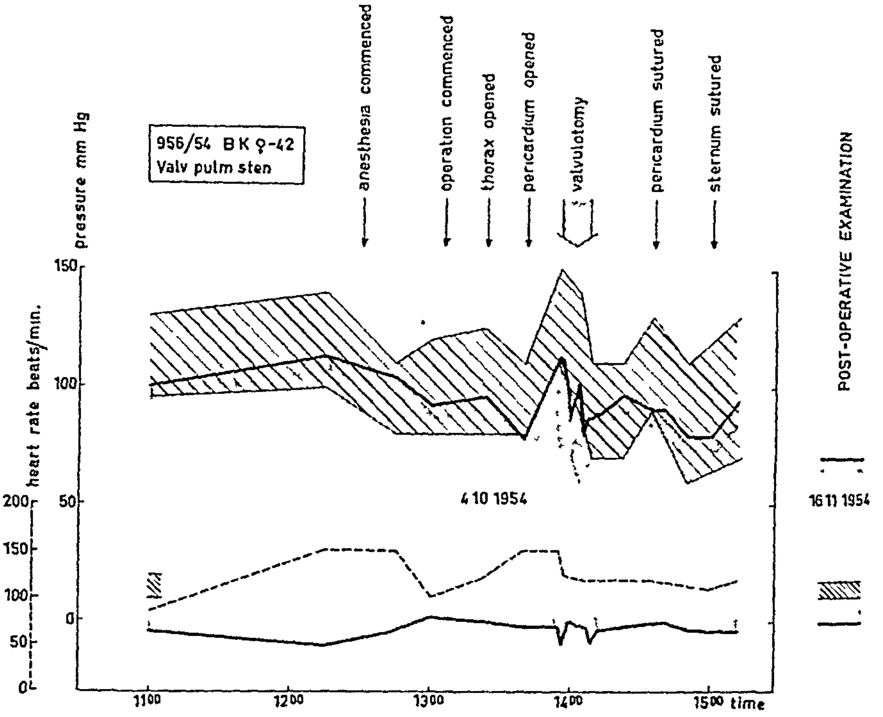

Fig. 2.

(An isolated pressure from the pulmonary artery is marked in the figure. This pressure was obtained via a catheter introduced by the operator into the pulmonary artery via the incision in the ventricular wall.)

Fig. 2 shows variations in pressure in the right ventricle in a 12-year-old girl operated upon for valvular pulmonic stenosis, ad modum Brock. The pressure values here show *lower* values *before* the valvulotomy than after the operation. As in Fig. 1, the pressure obtained on postoperative examination in the waking state is lower than any pressure recorded during the operation.

Fig. 3 illustrates the variations in pressure in the pulmonary artery during valvulotomy performed on a 4-year-old boy. A slight defect in the septum of the ventricle had been diagnosed preoperatively; after the splitting of the valve it was decided that the defect in the ventricular septum must be of considerable size, so that the effect of the valvulotomy was not favorable. During the final hour after the clamping, but rose again to show on postoperative examination 3 months later that there is still a certain hypertension in the pulmonary circulation

(For technical reasons, intracardiac pressure recordings were not made during the period 16 05 16 40 [see Fig 4])

In Figs 1 4, the right ventricle or the right ventricle and the pulmonary artery show a hypertension By way of comparison, pressure recordings during operation were made from a pulmonary artery in which the pressures were not so high This is shown in Fig 5 The patient was a 13 year old boy with coarctation of the aorta at the typical point below the branching off of the left subclavian artery

The variations in pressure in the pulmonary circulation were moderate, a reduction was noted during the narcosis, a rise during the manipulations of heart and vessels, although not to the same extent as the changes in the peripheral pressure during the clamping of the aorta

(As palpation of the radial pulse was applied in this case, it has only been possible to give the systolic value of the peripheral blood pressure)

DISCUSSION

What gave rise to this investigation was the possibility of being able, by means of a continuous recording of pressures during valvulotomy, to read the effect of the operation more precisely than has been possible through isolated pressure measurements via punctures of the right ventricle wall

The investigations presented here show that if, in these cases, isolated pressure measurements had been recorded, representative values would not have been obtained during the actual operation Altogether, the method of isolated pressure measurement—even under relatively standardized conditions—must be regarded as a rather unsatisfactory mirror of the intracardiac hemodynamic state[6]

By comparing the pressures recorded during operation with those recorded at a postoperative examination we have, moreover, been able to show that, in cases of pulmonary stenosis, the effect of the valvulotomy can only be judged at a control examination performed a considerable time after the operation This has been pointed out by several writers,[7, 8 9] but is at variance with what has been adduced in other quarters[5] Nor are the pressure values from the pulmonary circulation (right ventricle) immediately after the closure of a large potent ductus (Fig 4) valid at a postoperative examination 3 months later

In operations of aortic stenosis, Brock[3] has for diagnostic purposes found withdrawal curves from the aorta during actual operation very useful Isolated simultaneous pressures from the aorta and left ventricle immediately before and after valvulotomy are considered by Brock[3] and Bakst and his associates[1] to give a reliable notion of the result of the operation, although Brock makes the reservation that pressures during operation cannot be compared with those from a later occasion stage of the operation, the catheter was withdrawn to the right ventricle, so that values from this chamber were obtained during a short period. The recordings from the postoperative examination show diverging values as compared with those from the operation itself.

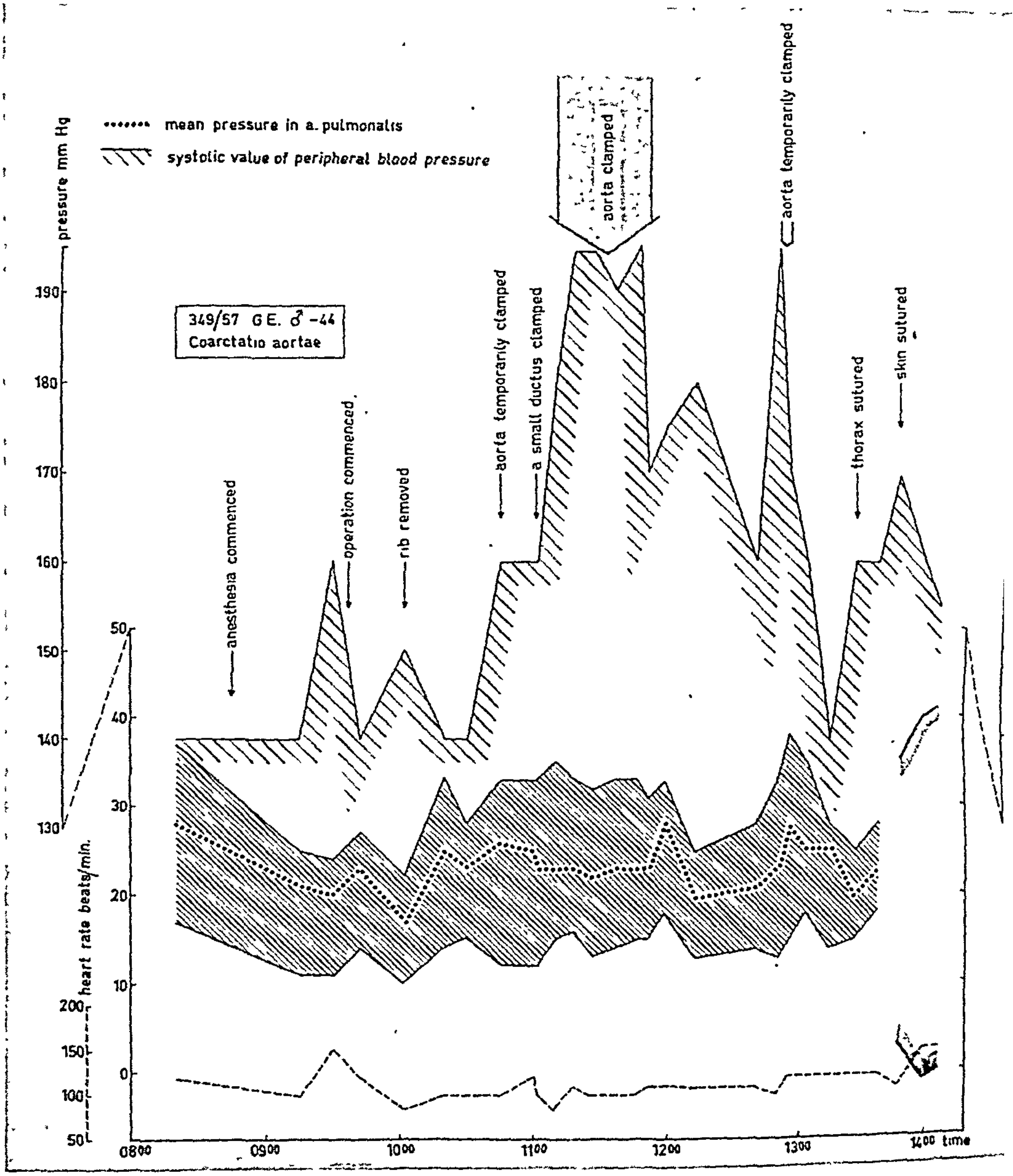

Fig. 5.

Fig. 4 shows the pressure values from the pulmonary artery and the rig ventricle during an operation performed on a 14-year-old boy with an atypic patent ductus. When the ductus had been clamped, the catheter was draw down from the right lateral branch of the pulmonary artery to the rig ventricle. The pressure rose during the dissection and the manipulation heart and vessels, fell after the clamping, appeared almost normalized about o

PRESSURE FLOW RELATIONSHIPS OF THE SIGMAMOTOR PUMP AS USED IN CARDIAC BYPASS

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THE development and clinical application of open heart surgery utilizing extracorporeal circulation has led to wide use of the Sigmamotor† finger type pump[1] As an occlusive type pump, it is designed to provide a constant output despite changes in the resistance against which it is pumping It has been assumed, then, that the pump output during cardiac bypass when the mean systemic pressure may be as high as 175 mm Hg will be the same as the predetermined flow during calibration against zero pressure[2] In our early experience with this pump in the laboratory, it was found that the actual output during cardiac bypass was less than the previously calibrated flow, resulting in excessive dog mortality Subsequent studies revealed a sharp drop in pump output with increased pressure unless extreme care was taken to obtain an optimum occlusive setting of the pressure plate The pressure flow characteristics of this pump with and without the optimum occlusive setting and a method for obtaining the optimum occlusive setting constitute the subject of this report

METHODS

A Sigmamotor Model T 6S cross circulation pump of a capacity of 4,500 c c per minute was used The calibrations were performed using a 20 cm long, ¾ inch I D gum rubber tube in the pump head, which was connected distally to a 5/16 inch I D plastic tube of 80 cm in length and proximally to a similar plastic tube which included a Monel metal filter ‡

The level of the perfusing solution in the reservoir was maintained at approximately the same height as the pump head A catheter was attached to the end of the distal limb by a tapered plastic adapter The catheter varied in size according to the anticipated caliber of the artery to be cannulated For these experiments the size was a No 12 or No 14 Fr The occlusive setting of the pump was determined by elevating the distal plastic tube 2 or more feet above the pump head and adjusting the pressure plate to that position which

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‡Kindly supplied by Abbott Laboratories North Chicago Ill

SUMMARY

Intracardiac pressure measurement, with the help of a catheter introduced into the heart before the beginning of the operation, is described in 3 cases of valvular pulmonary stenosis, 1 case of atypical patent ductus arteriosus, and 1 case of coarctation of the aorta. Continuous pressure recording has in this way been rendered possible during actual valvulotomy of the pulmonary valves, during clamping of a wide ductus, and in connection with clamping of the aorta during operation for coarctation of the aorta.

The advantage of this examination method as compared with the isolated pressure recordings via punctures of the heart cavity and vessels is pointed out.

The effect of valvulotomy in cases of pulmonary stenosis can only be assessed through preoperative and postoperative pressure measurements in the right ventricle, which is illustrated by comparing values from the actual operation with those obtained on postoperative examination, at least one month later.

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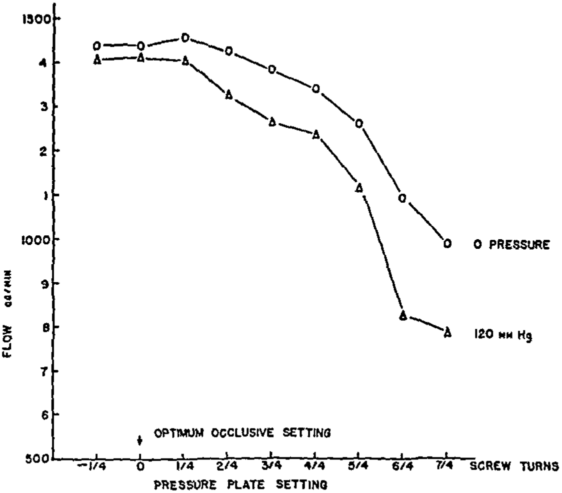

Fig 2.—Flow With Varied Setting. The effect of peripheral pressure on pump output with varying degrees of nonocclusiveness as quantitated by ¼ turns of the pressure plate screws

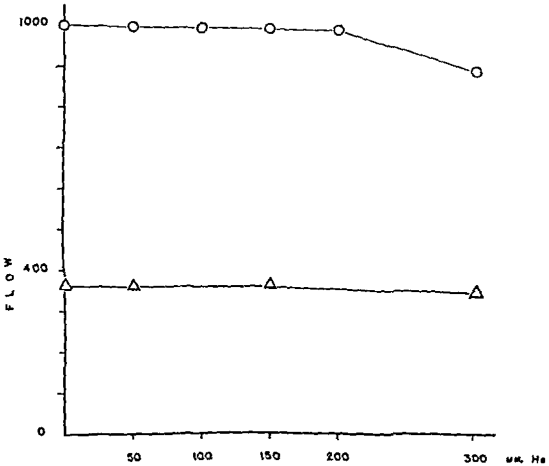

Fig 3.—Optimum Occlusive Setting. Negligible effect of pressure on pump output with optimum occlusive setting

prevented regurgitation of the fluid through a complete cycle of the pump. When this position of the pressure plate had been determined by the maneuver described, each pressure plate screw was loosened by ¼ turn, and this setting was taken to be the optimum occlusive setting.

RESULTS

The effect of pressure on the output of the pump with a deliberately nonocclusive setting of the pressure plate for flows of from 360 c.c. per minute to 980 c.c. per minute is shown in Fig. 1. It is apparent that with an average mean systemic blood pressure of 100 mm. Hg, reductions of 60 per cent from the calibrated flow against zero pressure can occur.

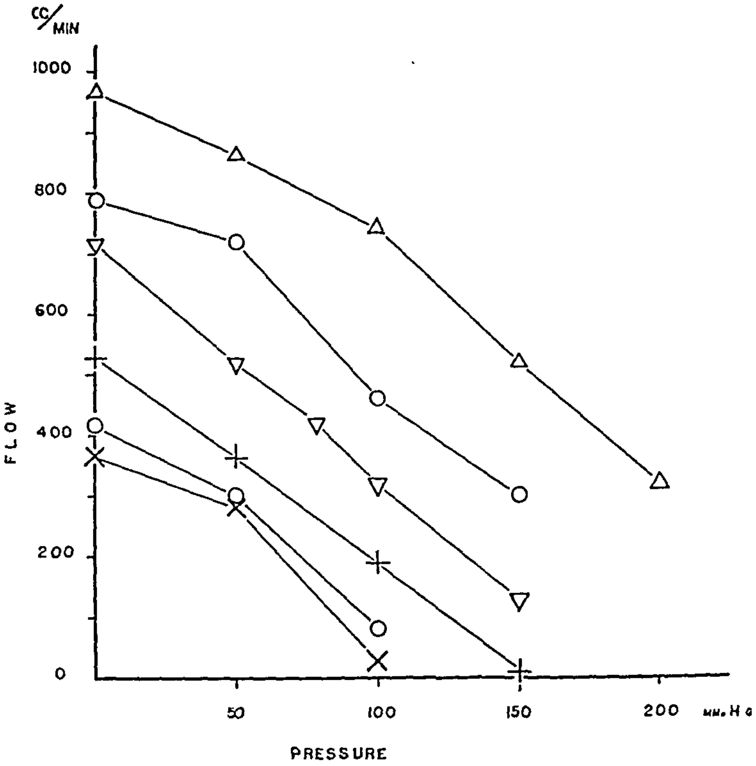

Fig. 1.—Nonocclusive Setting. The effect of pressure on pump output with a deliberately nonocclusive setting (see text for discussion.)

The effect of pressure on flow with varying degrees of nonocclusiveness as quantitated by ¼ screw turns from the optimum plate setting is shown in Fig. 2. These data indicate that minor degrees of deviation from the optimum occlusive setting can have a significant effect on pump output and that this effect is magnified by increasing pressure. With optimum occlusive setting of the pressure plate there is a negligible effect of increasing pressure up to 240 mm. Hg on flow as is demonstrated in Fig. 3.

DISCUSSION

The Sigmamotor pump, designed to operate as an occlusive pump, can be relied upon to maintain the predetermined output against increasing pressure

RESISTANCE TO CATHETER PUNCTURE OF VARIOUS CARDIOVASCULAR STRUCTURES

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THERE are very few untoward incidents or fatal results related to cardiac catheterization despite its wide use in the investigation of heart disease In particular, puncture of cardiac chambers or intrathoracic vessels has rarely been reported[1] Some canine physiologic studies were being made in this laboratory which involved the insertion of a catheter through a brachiocephalic artery In a series of 11 dogs, 3 suddenly developed shock and eventually died Viewed fluoroscopically, the catheter appeared to be free within the chest cavity Thoracocentesis was performed, and pure blood was obtained Autopsy in each instance showed a minute puncture at the origin of one of the brachiocephalic arteries from the aortic arch These incidents aroused interest as to how much stress would puncture the great vessels of the cardiovascular system Haddy and his co workers[2] found a 10 per cent mortality in left heart catheterization in dogs, which included shearing of the brachiocephalic artery from the aorta, and perforation of the aorta and of the left ventricle, while none occurred with right heart catheterization Several studies have been made on the tensile strength of strips or cylindric segments of blood vessels,[3-10] but none on resistance to impact stress on a sharply localized area of a vessel as might be made by the thrust of a cardiac catheter A simple method of carrying out such a study was therefore devised

MATERIALS

A Lucite platform was constructed (*A*) in Fig 1, with a 4 mm hole in the top plate Another piece of Lucite (*B*) with a corresponding hole was placed over the platform, and over this hole was attached a piece of glass tubing (*C*), 2 cm long and of 0 5 cm internal diameter Clamps (*D*) were provided to permit compression between the plates Pieces (*E*) about 4 cm long were cut off from the tips of Nos 5 and 8 Goodale Lubin catheters A piece of rigid stainless steel wire (*F*), corresponding to the internal diameter of each catheter tip, was attached to a small thin piece of balsa wood (*G*) This could then be threaded through the corresponding catheter segment to provide rigidity The wire length was adjusted so that the tip fell about 1 mm short of the tip

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*Research Fellow of the East Central Indiana Heart Foundation only if the optimum occlusive setting of the pressure plate is verified prior to each use of the pump. Minor deviations from this optimum setting can result in significant reduction in flow when pumping against systemic pressure. In a study of the physiologic responses during cardiac bypass, mean perfusion pressures of 34 to 175 mm. Hg were reported from this laboratory.[3, 4] It is apparent, then, that during cardiac bypass when systemic pressures of this magnitude are encountered, the pump output may be reduced 5 to 60 per cent from the previously calibrated output against zero pressure if care is not taken in obtaining the correct pump setting. Such reductions in pump output may contribute to the acidosis, hypoxia, and death due to low flow, when it is thought that adequate flow is being utilized.

SUMMARY

The pressure-flow characteristics of the Sigmamotor pump with and without optimum occlusive setting of the pressure plates are presented. The necessity for obtaining the optimum occlusive setting to assure the predetermined output of the pump during cardiac bypass is emphasized. A method for verifying the optimum occlusive setting of the pressure plate is described.

ADDENDUM

Sigmamotor, Inc., has recently developed and made available a new pump head with vernier knob adjustment of the pressure plate. Thus, the need for individual adjustment of the pressure plate screws is eliminated. However, the necessity for optimum occlusive setting of the pressure plate remains of utmost importance.

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platform, and the 2 plates were clamped together to hold the tissue in place (*K* in Fig. 1), without placing it on stretch. The catheter tip assembly was placed through glass tube (*C*) with its end supported by the tissue. The glass tube (*H*) was placed on the balsa plate of the catheter tip and allowed to rest freely. Lead weights were carefully dropped into the tube from a fixed point at the top, starting with the 5 Gm. weight, and progressing to the next higher weight until the tissue was punctured. Each weight was removed from the tube before dropping the next one. The piece of glass tubing attached to the upper piece of Lucite supported the catheter tip so that it did not bend when a weight was dropped. The vessel was therefore subjected to a fixed static load of the combined weights of the glass tube and catheter set-up resting on the tissue (approximately 80 Gm.), plus the impact load of the graduated weights dropped through a fixed distance. Pilot experiments showed that additional static loading of approximately 1,600 Gm. or 20 times the static loads applied in the apparatus were necessary to produce rupture of these vessels held in this manner. Thus, the static loads used in the experiment were of minor significance in comparison to the impact loads. With distance held constant, the weights necessary to produce rupture on impact should constitute reliable indices of relative resistance to puncture.

The superior vena cava, main pulmonary artery, and aortic arch from one dog were tested. Determinations were also made at the origin of the brachiocephalic arteries from the aortic arch from this dog, as well as from 3 other dogs. All tissues were kept wet with normal saline solution throughout the experiment.

RESULTS

The results of 30 determinations on each great vessel of 1 dog, and also on a number of points along the aortic arches of 3 other dogs are shown in Tables I, II, and III.

DISCUSSION

Very few animals were used, for it was not the purpose to establish absolute values in dogs, but rather to compare tissue strength. Furthermore, it was expected that the possible effects on tissue structure of factors such as age, size, diet, and habits of the animal would exert the same influence on each determination of tissue strength. The significance of mean differences in puncturing weights of the different vessels were tested for by computing t ratios.[11]

As indicated in Table I, the difference in strength of the superior vena cava and the aorta was small. Nevertheless, the difference was sufficient to have occurred only 5 to 10 per cent of the time on the basis of chance. The results, then, suggest a possibility that the resistance to puncture of the superior vena cava is slightly greater than that of the aorta. The aorta and the superior vena cava were over 1.5 times as strong as the main pulmonary artery, a difference shown at a very high level of significance. It took a greater amount of weight to puncture the aortic arch with a No. 8 catheter than with a No. 5 catheter as might be expected with the force being resisted by a larger area of vessel in the case of the No. 8 catheter. There was a significant weakness at of the catheter. The steel wire and its balsa plate with a No. 8 catheter tip weighed about 1.2 Gm., and with a No. 5 catheter tip, 0.7 Gm. A glass tube (*H*), 30 cm. long, 2.2 cm. internal diameter, and weighing 78 Gm., with a rigid detachable bottom (a pill-bottle cap [*I*] over which a flat piece of balsa wood [*J*] was attached), was allowed to rest on the plate holding the catheter, and kept upright by loose encirclement with ring clamps. Lead weights of graduated sizes at 5 Gm. intervals, over the range from 5 to 45 Gm., were obtained.

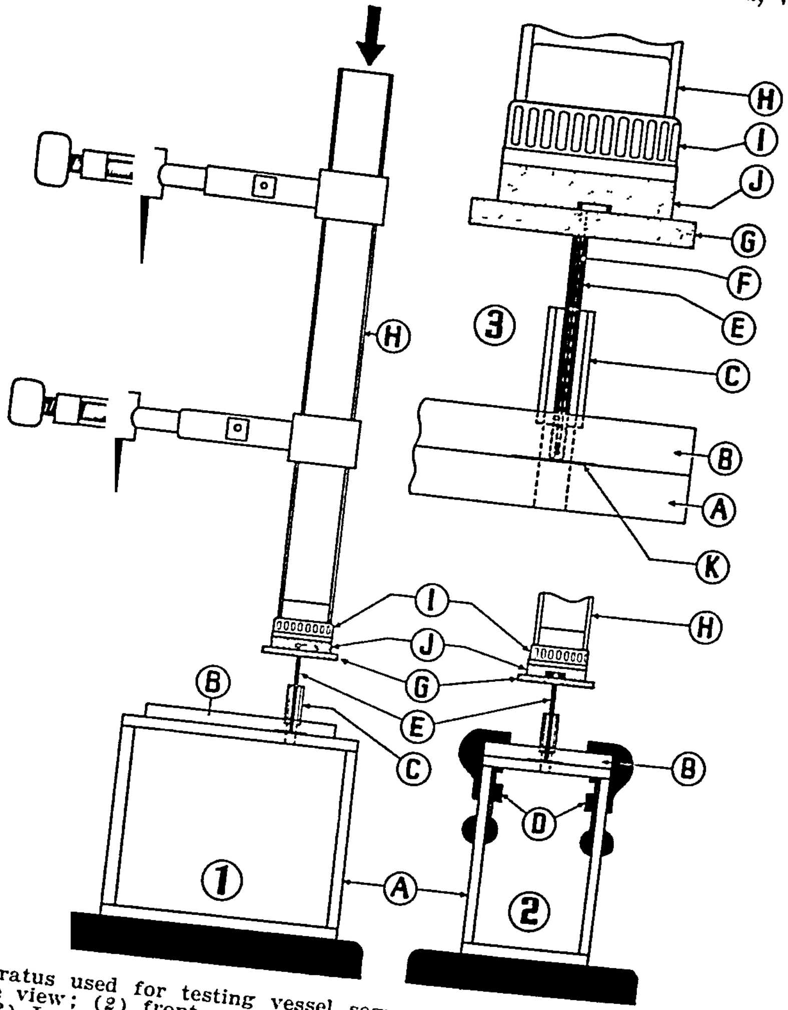

Fig. 1.—Apparatus used for testing vessel segments for relative resistance to catheter puncture. (*1*) side view; (*2*) front view, (*3*) detail of catheter tip resting on tissue. (*A*) Lucite platform, (*B*) Lucite top plate, (*C*) glass tubing, (*D*) clamps, (*E*) catheter tip, (*F*) steel wire, (*G*) balsa plate, (*H*) glass tube, (*I*) pill-bottle cap, (*J*) balsa wood attached to cap, (*K*) tissue.

The aortic arch, superior vena cava, and main pulmonary artery were sectioned from a large mongrel dog, weighing 31 pounds. Aortic arches were also obtained from 3 other dogs of various sizes.

METHOD

A segment of blood vessel was carefully split open and placed across the hole on the Lucite platform. The other piece of Lucite was laid across the

4. More stress was needed to puncture the aortic arch with a No. 8 catheter than with a No. 5 catheter.

We gratefully acknowledge the expert statistical advice of Dr. Ralph Reitan, Associate Professor of Psychology, Indiana University School of Medicine.

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the points where the brachiocephalic arteries originated from the aortic arch, especially at the proximal curve (Tables II and III). Pathologic changes have been found to occur more where the branches leave the aorta.[12] Since the most rapid flow occurs along the shortest course at a ramification, the proximal angle of a branching is exposed to more mechanical trauma than the distal angle. In this study the proximal curve of the right brachiocephalic artery as it came off the aortic arch was compared with the area between the two brachiocephalic arteries. The distal curve of the left brachiocephalic artery was not evaluated for comparative study because of the fewer determinations made at this point.

TABLE I*

	SUPERIOR VENA CAVA	MAIN PULMONARY ARTERY	AORTIC ARCH	AORTIC ARCH (NO. 8 CATHETER)
Mean	17.66	10.50	16.33	23.83
S.D.	2.22	1.35	2.28	2.76
Range	15-20	10-15	10-20	20-30
N	30	30	30	30

TABLE II*

	ORIGIN OF BRACHIOCEPHALIC ARTERIES				
	PROXIMAL	DISTAL	BETWEEN BRANCHES	TOTAL	AORTIC ARCH
Mean	11.36	14.28	14.58	13.33	15.33
S.D.	2.16		2.32	2.67	1.59
Range	10-15	10-20	10-20	10-20	10-20
N	11	7	12	30	30

TABLE III*

TISSUES UNDER COMPARISON	DIFFERENCE OF MEANS	t	p
Superior vena cava and main pulmonary artery	7.16	13.04	$<.001$
Superior vena cava and aortic arch	1.33	1.97	$<.10;>.05$
Main pulmonary artery and aortic arch	5.83	10.50	$<.001$
Aortic arch with Nos. 5 and 8 catheters	7.50	10.00	$<.001$
Total points of origin of branch arteries and aortic arch	2.00	3.08	$<.01$
Origin of branches: at proximal curve and between branches	3.22	3.16	$<.01$

*All weights are expressed in grams and refer to weight dropped through a constant distance in the described apparatus which produced rupture of the tissue. N = number of determinations. S.D. = standard deviation.

All determinations were made with a No. 5 catheter unless otherwise stated.

SUMMARY

1. A simple method was devised for testing comparative tissue resistance to catheter puncture stress in the main pulmonary artery, superior vena cava, aortic arch, and points of origin of the brachiocephalic arteries in dogs.

2. The results suggested slightly increased tissue resistance in the superior vena cava as compared with the aortic arch. The main pulmonary artery was weaker compared with these two vessels.

3. There was a significant weakness at the points where the brachiocephalic arteries left the aortic arch, especially at the proximal curve, compared with the aortic arch.

recorded The periaortic [illegible] of the as[illegible]ding aorta. [illegible] at its ori[illegible] The post[illegible] the junc[illegible] of right atri[illegible] A partia[illegible] clusion clamp [illegible] approxi[illegible]ely 3 cm. dis[illegible] [illegible]

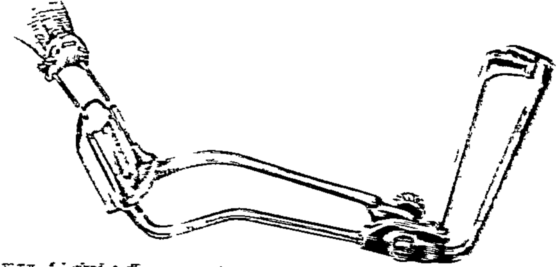

Fig. 2.— [illegible]

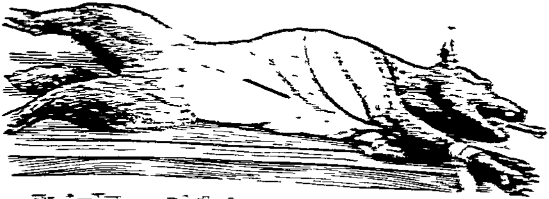

Fig. 3.— [illegible]

[illegible] is made in the [illegible] [illegible] sutured to the edges [illegible] The pouch is [illegible] [illegible]

[illegible]

DIRECT VISION APPROACH TO THE AORTIC VALVE WITHOUT OCCLUSION OR BYPASS

Howard L. Gadboys, M.D.,* James L. Harrison, M.D.,** and
Robert S. Litwak, M.D.***

INTRODUCTION

A TECHNIQUE is presented which permits exposure of an aortic cusp through the sinus of Valsalva without circulatory occlusion or bypass. A clamp (Fig. 1)† consisting of two coapting baffles, one fenestrated and the other recessed, is positioned over the noncoronary aortic cusp to form a chamber in which direct visualization and manipulation of the cusp are possible. Tailoring of all or part of the cusp and replacement by a graft or prosthesis have been carried out on experimental animals. The operating time within the valve ring is relatively unlimited since neither extracorporeal circulation nor vascular occlusion is required.

PROCEDURE

One hundred and three adult mongrel dogs, weighing 12 to 28 Kg., have been operated on. Intravenous Nembutal anesthesia is administered, an endotracheal tube inserted, and ventilation maintained with an Emerson, pressure-sensitive, cycling respirator. The inspiratory pressure varies from +14 to +18 cm. H_2O and the expiratory pressure from +4 to +6 cm. H_2O. After positioning the dog for a right thoracotomy (Fig. 2), a control electrocardiogram is taken and the cardiogram is monitored during the procedure with a Sanborn oscilloscope. A small midline incision is made and the xyphoid is resected in the animals having autogenous graft valvular replacement. The xyphoid is tailored to form a cartilaginous frame. This incision is closed and a standard intercostal incision is made in the right third interspace. A modified Finochietto retractor is inserted. The right lung is retracted caudally to expose the base of the heart. A 3 by 4 cm. segment of pericardium (Fig. 3) is removed, defatted, and the free valvular graft is constructed. A pericardial envelope is made, inverted to place the suture line inside, and the tailored xyphoid stent is inserted. The flap is immersed in dilute heparin solution until needed. Following re-expansion of the lungs, the right lung is again retracted caudally with a moist sponge. A traction suture in the tip of the right atrial appendage is used to retract the appendage, exposing the base of the aorta (Fig. 4). Control aortic and left ventricular pressure pulses are then

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†The baffle clamp was constructed by Codman and Shurtleff, Inc., 104 Brookline Ave., Boston, Mass.

Pressure measurements are made with Statham strain gauges (P23A-P23D) connected to a Sanborn 150 M 4 channel recorder monitored with a Sanborn Visoscope The manometers are all balanced to the same fluid level arbitrarily set at table top height[3, 7]

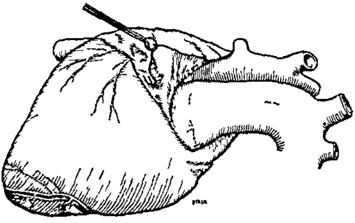
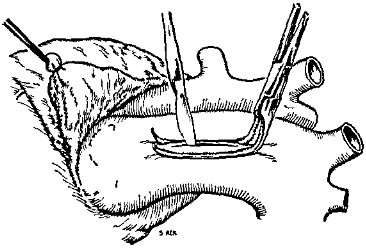


Fig 4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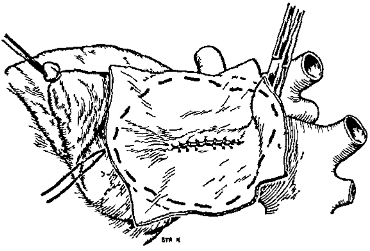
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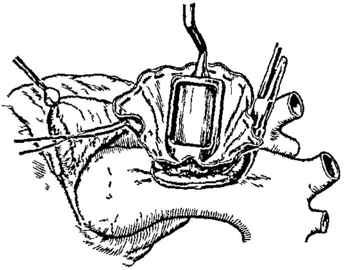
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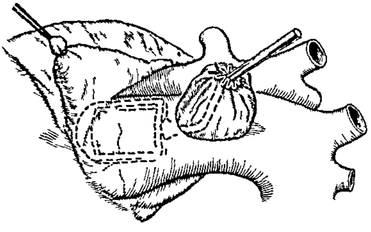


Fig 8

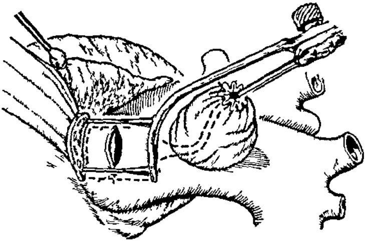


Fig 9

Fig 4—Posterior sinus of Valsalva exposed by traction on right atrial appendage Line of distal aortic incision is shown

Fig 5—Partial occlusion clamp applied to aorta and distal incision made

Fig 6—Pericardial or plastic envelope sutured to edges of incision

Fig 7—Recessed baffle inserted into pouch

Fig 8—Purse string suture tied Recessed baffle advanced to region of posterior sinus of Valsalva

Fig 9—Fenestrated baffle applied positioned over aortic sinus and clamp locked in place Transverse incision made over lunular edge of posterior (noncoronary) aortic cusp

sutures are loosely tied. A second layer of mattressed adventitial sutures is then taken. Pressure is applied to the suture line for 10 minutes as the baffle clamp is unlocked. The unlocked clamp is dismantled, and the recessed baffle is returned to the pouch, permitting the graft to flap between the remaining leaflets and the aortic wall (Fig. 11). The pouch is excluded by replacing the partial occlusion clamp to permit its removal. The distal aortic incision is closed with mattress sutures and oversewn. Pressure measurements are again recorded.

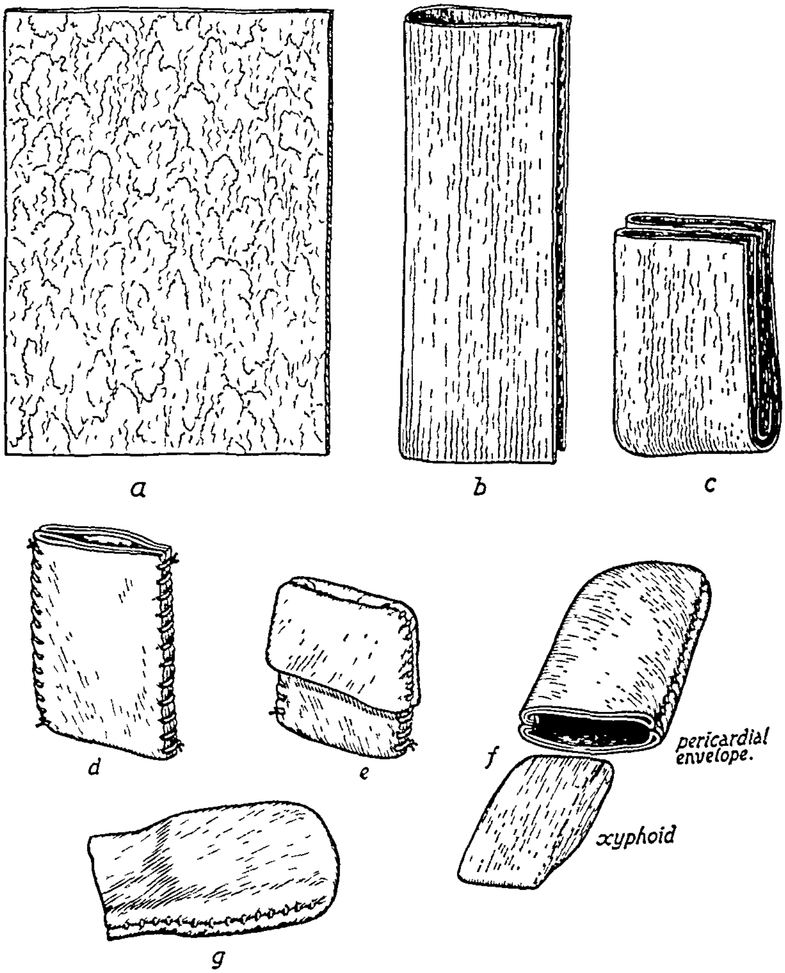


Fig. 3.—Preparation of free autogenous flap from pericardium and xyphoid. Pericardial envelope is inverted to place suture line inside.

The pericardium is ordinarily not approximated. The chest is closed over an intercostal tube connected to an underwater seal. The lungs are expanded and the intercostal tube is removed. Spontaneous respirations return within seconds of disconnecting the respirator. The incisions are coated with collodion, another electrocardiogram is recorded, and the endotracheal tube is removed.

Postoperatively, the dogs receive intramuscular penicillin for 7 days. There is no restriction of activity postoperatively and the dogs are not digitalized.

normothermic. Bradycardia followed the production of a satisfactory S-A nodal block, but the subsequent response of the animals to even moderate hemorrhage was catastrophic. We could not help but feel that blockade of the sino-atrial node in normothermic animals served to rob them of an effective defense mechanism against shock.

RESULTS AND COMPLICATIONS

Survivals have been followed for periods up to 16 months. The mortality statistics cannot be evaluated as a group since several technical modifications have been made. The last 10 dogs were operated on by the technique described with one operative fatality (10 per cent). This dog had electrocardiographic evidence of an anterior myocardial infarction. The oscilloscopic tracing revealed S-T segment displacement as the posterior cusp was being resected. This rapidly progressed to ventricular fibrillation and death. The animal was autopsied with the baffle clamp still locked in place. There was no encroachment on the coronary ostia by the clamp, and dissection of the coronary arteries and myocardium was uninformative. The dog had had moderately profuse hemorrhage from the pericardial pouch with no blood replacement.

In contrast to this group, there were 10 operative deaths in the first 20 dogs operated on by a similar but less refined technique (50 per cent). Hemorrhage was the precipitating factor in 7 of these. Four of the 7 hemorrhagic deaths were due to faulty construction of the baffle clamp. There was tearing of the aorta from sharp angles present on the original clamp. Utilization of an inadequate aortic pouch caused 2 deaths in this group, and unsatisfactory hemostasis of the aortic sinus suture line led to the demise of the third. Care is now taken to avoid even slight tension of the proximal aortic incision to prevent intimal tears when the baffle clamp is released. One dog developed ventricular tachycardia following cardiac manipulation. Pronestyl was administered but a 3 1 A-V block supervened. This progressed to fibrillation which could [illegible] be reverted electrically. Another dog died following inadvertent transfusion of blood containing 75 ml. of sodium citrate solution after closure of the [illegible] One dog had marked electrocardiographic changes at the completion [illegible] procedure suggesting an acute myocardial infarction. No gross or microscopic pathologic changes were found.

Several complications of the entire group of animals warrant consideration. Biochemical analyses during the earlier procedures revealed acidosis in many of the dogs with blood pH ranging to 7.13. This was shown to be respiratory acidosis precipitated by a subatmospheric expiratory phase of the respirator Adjustment of the respirator to maintain positive pressure cycling from +16 cm H_2O to +4 H_2O at 20 to 30 excursions per minute has corrected this abnormality.

There have been 5 late postoperative deaths in the 103 animals in which bronchopleural fistula was either a major or contributing factor. The fistulas originated in pulmonary parenchymal tears sustained at operation. Five dogs died from empyema. Embolization of the flap-valve occurred in [illegible] s who

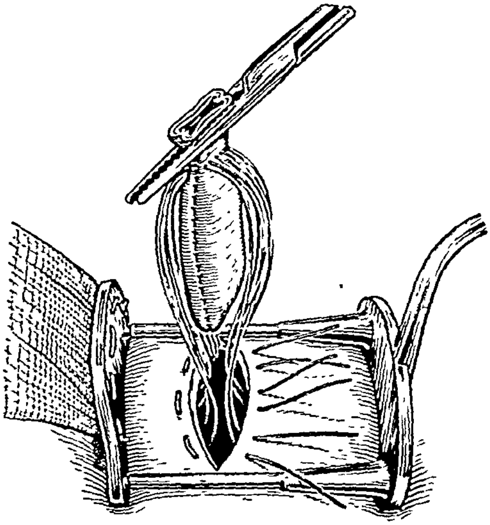


Fig. 10.

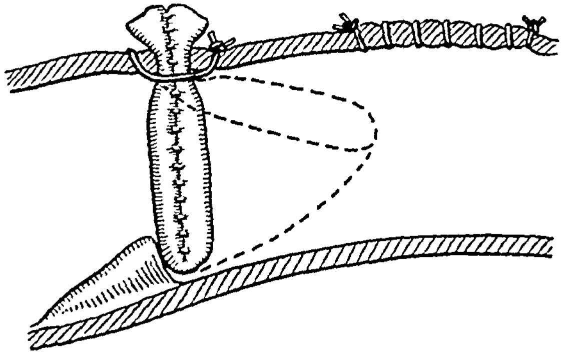


Fig. 11.

Fig. 10.—Sutures placed in aorta and free graft before advancing graft into chamber.
Fig. 11.—Graft swings free with systole and engages remaining cusps during diastole.

TECHNICAL VARIATIONS

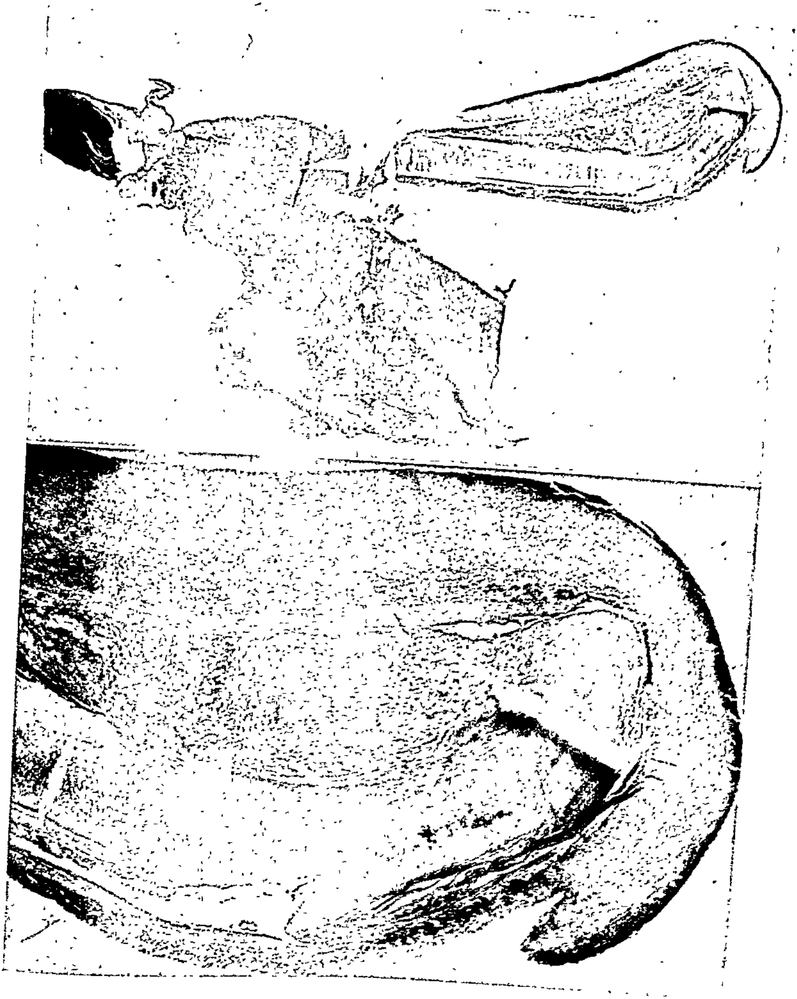
The experiment has been set up to permit evaluation of the regurgitation produced and the effectiveness of the corrective measures. A control series of animals has had cusp resection only. Another group has had cusp resection followed by direct vision replacement with a graft or prosthesis. To date, our primary interest has been cusp replacement with an autogenous tissue graft. Pedicled pericardial grafts with cartilaginous stents theoretically seemed to offer greater promise of survival because of the retained blood supply. The theoretical advantage was decidedly outweighed by the greater technical ease with which the free grafts could be handled. There has not yet appeared to be any significant benefit from maintaining the blood supply. Woven nylon fabric has been used both to buttress the suture line and to replace the pericardium. The tendency of the fabric to fray under tension, even when the edges have been heat fused, has limited its usefulness.

The problem of operative hemorrhage prompted us to use both hypothermic and hypotensive anesthesia to minimize bleeding. Hypothermia, in this limited experience, did not cause significantly great falls in blood pressure at the therapeutic range (28° to 31° C.) to be of help. Considerable decrease in aortic tone and hemorrhage was obtained by using a hypotensive drug (Thiophanium). The dogs were extremely sensitive to the drug, however, and total doses of as little as 0.5 mg. produced profound and irreversible hypotension in 3 of 8 large dogs. Fractional injection and continuous intravenous drip of extremely dilute solutions were tried but the results were unpredictable. Reversal of the precipitous hypotensive effects by peripheral vasoconstrictors did not materially alter survival.

Although most of the animals were anesthetized with intravenous Nembutal (25 mg./Kg.), Vinethene-ether anesthesia was also employed.

On occasions, when manipulation of the right atrium produced arrhythmias, procaine was infiltrated into the sino-atrial node. These dogs were

A.



B.

Fig. 13.—A. Low-power photomicrograph of graft in animal sacrificed 2 weeks postoperatively. Organized thrombus present on proximal side.

B, Higher power photomicrograph of graft.

died 8 days and 2½ months following surgery. One operative fatality resulted from embolization to the brain and aortic trifurcation of a thrombus formed in the aortic pouch.

Pulsus alternans has been noted during operation in approximately 5 per cent of the procedures. The etiology has not been determined, but it has had no prognostic significance. This is in sharp contrast to the gravity of pulsus alternans clinically.

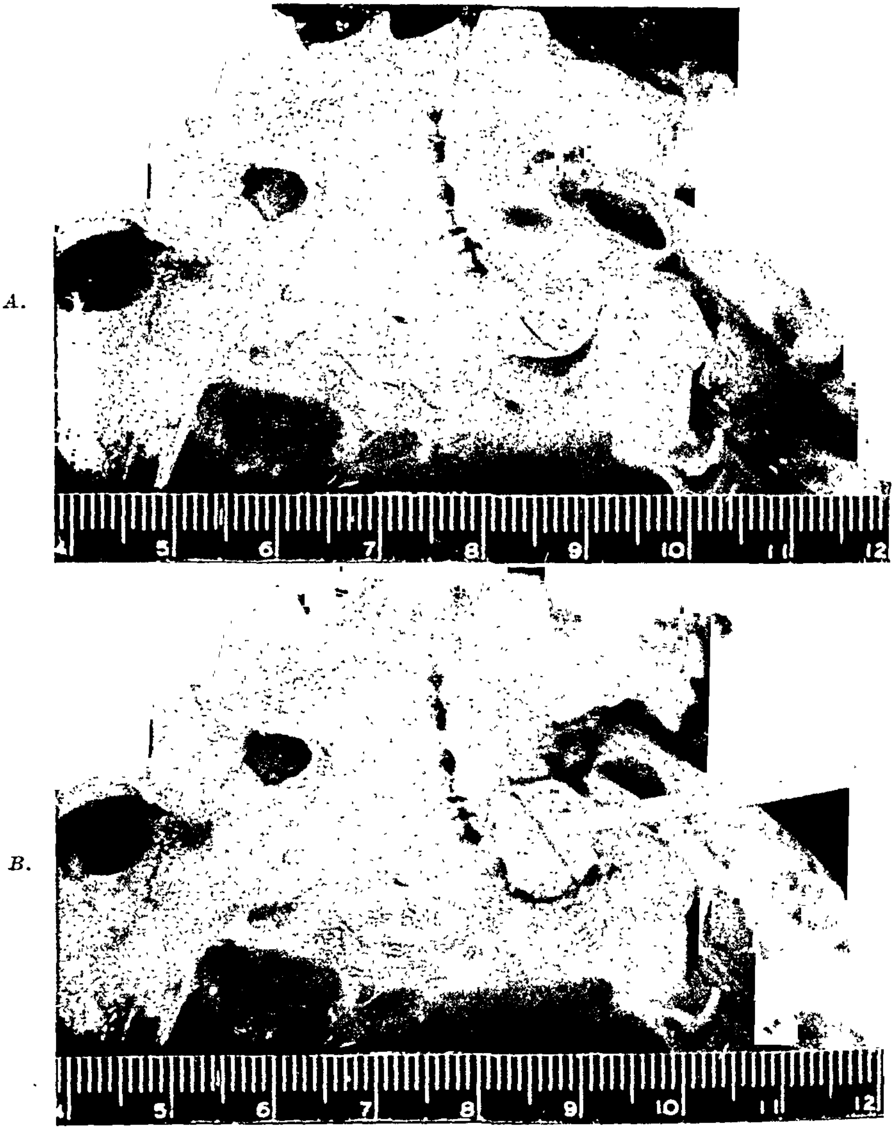


Fig. 12.—Appearance of free graft in animal sacrificed 11 weeks after excision of posterior cusp with autograft replacement. *A*, Position during diastole. *B*, Position during systole.

The fate of the autogenous flap valves is being studied. The animals have been followed for periods up to 16 months and are being sacrificed at calculated intervals (Figs. 12, 13). The principal postoperative difficulty encountered has been suture-line hemorrhage precipitated by intimal tears at the no attempt was made to restore blood pressures to preoperative levels (Fig 16) The animal in whom a twofold variation occurred (Feb 27, 1957) died on March 7, 1957 Autopsy revealed that the flap had embolized leaving pure aortic insufficiency The cause of death was pulmonary congestion and edema

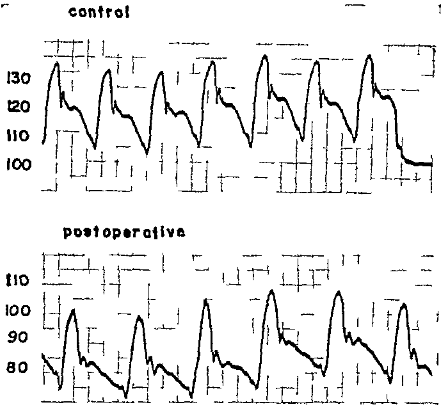


Fig 15—Aortic insufficiency with cusp replacement Aortic pressure tracings before valvular surgery and after production of aortic insufficiency with autogenous cusp replacement

TABLE I EXPERIMENTAL AORTIC INSUFFICIENCY*

	DATE	PROCEDURE	PREOPERATIVE AORTIC PULSE PRESSURE (MM HG)	POSTOPERATIVE AORTIC PULSE PRESSURE (MM HG)	FATE
1	Dec 27, 1956	Cusp resected	Aorta angulated inadvertently		Operative death
2	Jan 3, 1957	Cusp resected	Pressures not obtained		Died, Jan 7, 1957, pulmonary edema and congestion
3	Jan 7, 1957	Cusp resected	15	85	Died, Jan 18, 1957, pulmonary edema and congestion
4	Jan 8, 1957	Cusp resected	25	40	Died, March 21, 1957, pulmonary edema and congestion
5	Jan 10, 1957	Cusp resected	15	40	Died, Jan 10, 1957, pulmonary edema
6	Jan 11, 1957	Cusp resected	13	55	Died, Jan 25, 1957, cardiac tamponade
7	Jan 16, 1957	Cusp resected	10	43	Died, Feb 3, 1957, pulmonary edema and congestion
8	Jan 18, 1957	Cusp resected	13	60	Died, Jan 27, 1957, pulmonary edema and congestion
9	Jan 23, 1957	Cusp resected	7	28	Died, Feb 4, 1957, pulmonary edema and congestion
		Mean Pulse Pressure	14	50	

*Alterations of pulse pressure produced by resection of t erior (noncoron) aortic cusp proximal edge of the aortic sinus incision. This problem appears to have been obviated by eliminating suture-line tension. Prosthetic valves other than nylon-covered stents have not yet been evaluated.

Intracardiac operating times have varied from 30 minutes to 180 minutes with survival. The presence of the clamp is generally well tolerated if care is taken to avoid angulation of the ascending aorta. We have been able to demonstrate circumscribed areas of pressure necrosis from the clamp in only 2 animals. In each, incorrect application of the clamp on the base of the right atrium produced distortion.

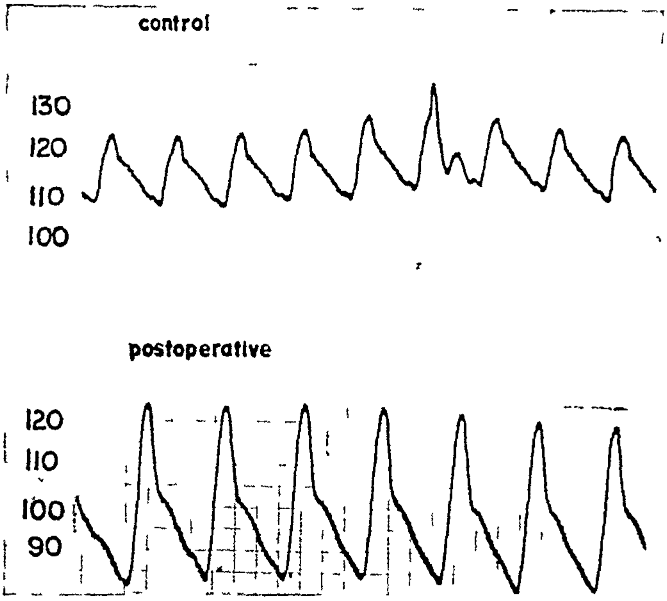


Fig 14.—Aortic insufficiency without cusp replacement Aortic pressure tracings before and after production of valvular regurgitation by partial excision of posterior cusp

HEMODYNAMIC OBSERVATIONS

Uncorrected aortic insufficiency was produced in 9 animals by partial or complete excision of the posterior (noncoronary) aortic leaflet (Fig. 14). Pulse pressures were increased an average of 4 times, the smallest increase changing from 25 mm. Hg to 40 mm. Hg and the largest deviation from 15 mm. Hg to 85 mm. Hg (Table I). The dogs were permitted moderate activity and were not digitalized. The longest survival was 10½ weeks. There was one operative fatality, one animal died from cardiac tamponade 2 weeks after surgery, and 7 dogs succumbed from pulmonary congestion and edema. At autopsy the 7 dogs manifested no evidence of cardiac dilatation or increased pericardial tension.

A completed series of dogs in whom cusp excision was followed by valvular replacement (Fig. 15) is not yet available since an acceptable mortality rate for this combined procedure has only recently been attained. The last 7 dogs in whom this technique was carried out can be compared with the 7 dogs in whom uncorrected aortic insufficiency was produced and pressure measurements obtained (Table II). No conclusions can be drawn from this series since

To date the only clinically acceptable technique for the treatment of aortic insufficiency has been the Hufnagel valve.[5] Any device inserted distal to the aortic valve ring, however, reduces the regurgitant work load at the expense of the diastolic phase of coronary arterial filling. It also requires an additional procedure if direct attack on the aortic valve is desired.

The ideal approach to the aortic valve, permitting direct vision for a prolonged time with insignificant mortality, has not been achieved. A bloodless chamber inserted without interruption of the circulation has given limited access to the aortic valve without limiting operating time. Exposure of the valve ring, moreover, permits placement of a graft or prosthesis at a level which will not affect coronary arterial flow. With the development of a satisfactorily durable graft or prosthesis, it should be possible to excise a central portion of the valve and utilize the remaining rim of calcific tissue to seat the corrective device.

SUMMARY

1. An experimental technique is described which permits direct visual approach to the aortic valve without circulatory occlusion or bypass. One hundred and three dogs have been operated upon.

2. The early operative mortality was 50 per cent. The present operative mortality is 10 per cent.

3. Intracardiac operating times with survival have varied from 30 to 180 minutes.

4. Pure aortic regurgitation has been produced by resection of all or part of the posterior (noncoronary) aortic cusp. No dog survived more than 10½ weeks.

5. Aortic regurgitation has been produced in a similar group of animals and simultaneously treated by insertion of a flap valve at the aortic ring. Survivals have been followed for periods up to 16 months.

6. The fate of autogenous flap valves and prostheses is being studied.

We wish to thank Dr. John J. Byrne, director of the Third (B.U.) Surgical Service and Research Laboratory, for his continued encouragement, Dr. James W. Dow and Dr. Eric Berglund for physiologic counselling, Mr. Joseph Ferrara and Misses Joan Hartigan and Paulette Burstein for their technical assistance, and Miss Violet Peters for the preparation of the manuscript.

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TABLE II. EXPERIMENTAL AORTIC INSUFFICIENCY WITH INSERTION OF FREE AUTOGENOUS FLAP VALVE*

DATE	PROCEDURE	PREOPERATIVE AORTIC PULSE PRESSURE (MM. HG)	POSTOPERATIVE AORTIC PULSE PRESSURE (MM. HG)
1. Jan. 29, 1957	Cusp resected and graft inserted	20	33
2. Feb. 11, 1957	Cusp resected and graft inserted	22	35
3. Feb. 19, 1957	Cusp resected and graft inserted	23	31
4. Feb. 27, 1957	Cusp resected and graft inserted	15	33
5. Mar. 5, 1957	Cusp resected and graft inserted	18	16
6. Apr. 8, 1957	Cusp resected and graft inserted	25	35
7. Apr. 25, 1957	Cusp resected and graft inserted	25	30
	Mean Pulse Pressure	21	30

*Pulse pressure alterations of last 7 dogs having cusp resection and replacement with free autogenous flap valve.

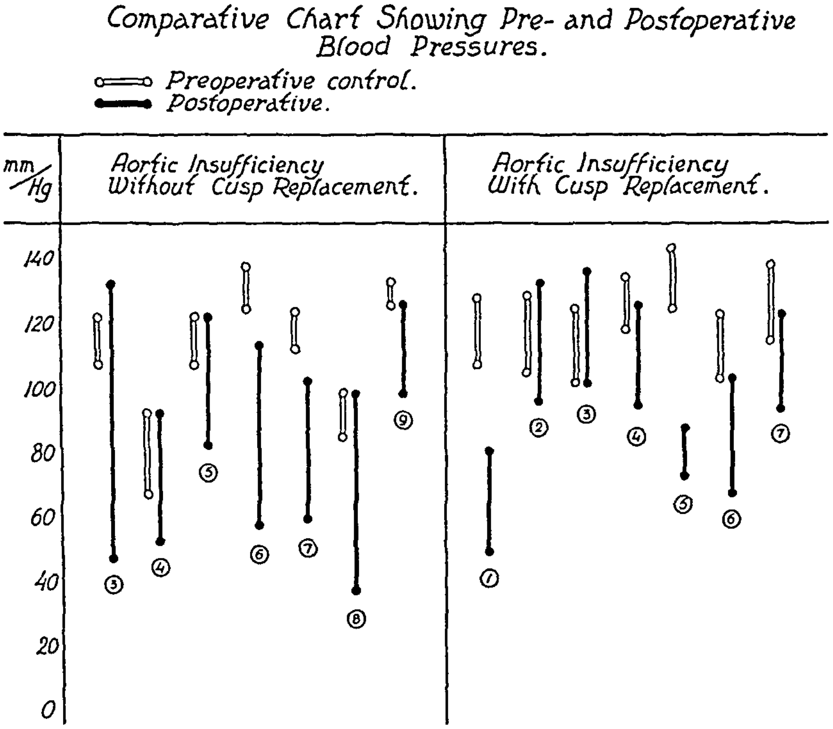

Fig. 16.—See text.

DISCUSSION

Naturally occurring aortic stenosis and aortic regurgitation are generally well tolerated for long periods of time, but both are serious lesions. It has been shown experimentally and clinically that acutely produced insufficiency is handled poorly since compensatory mechanisms cannot develop. The adherent but noncalcified stenotic aortic valve responds well to dilatation and separation of the commissures. The pathologic changes in a rigid, calcified valve, though, do not readily succumb to such treatment. Removal of a portion (⅓ or more) of the deformed valve restores normal hemodynamic relationships[2] but is not clinically feasible because of the more severe regurgitant lesion produced. Therefore, total successful restoration of the diseased aortic valve depends on a satisfactory method for controlling regurgitation.

The neoplasm was soft and pale red with scattered, dark bluish-black spots. The histologic diagnosis was leiomyosarcoma, of low-grade malignancy (Fig. 1). Only inflammatory changes were found in the enlarged lymph nodes.

Postoperatively, empyema developed in the left pleural cavity and required open drainage on April 28, 1936. The patient gradually improved after this and was discharged from the hospital on June 16, 1936.

During the next 15 years the patient considered himself well, but noticed an occasional discharge of suture material from the thoracic incision. When seen in 1953, he was working as an operator of a bulldozer. Localized empyema had developed with a long sinus tract which did not show any tracheobronchial communication. This sinus tract was unroofed and a Schede type of thoracoplasty was performed. Recovery was prompt and he returned to full time work. He has been seen intermittently since then. On April 5, 1957, he was well and working.

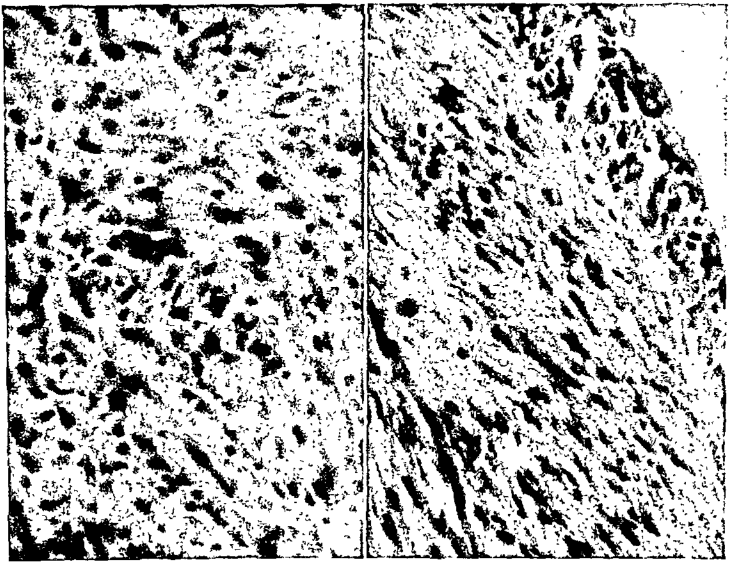

Fig. 1.—*A* and *B*, Photomicrographs of two regions of the leiomyosarcoma arising from the bronchus. (×430; reduced ⅝.)

DISCUSSION

Before the first successful pneumonectomy by Graham,[3] many surgeons were devising surgical techniques, performing pneumonectomies on experimental animals, and attempting resection of greater and greater portions of the lung. As early as 1920, Sauerbruch[9] had accomplished a lobectomy and, in 1931, Churchill[2] had resected two lobes of the right lung. In 1931, Nissen[6] had ligated the hilar structures of a bronchiectatic lung and it had subsequently sloughed out; a similar case was reported in 1933 by [illegible]

PNEUMONECTOMY FOR LEIOMYOSARCOMA

Patient Well 21 Years Later

Alton Ochsner, M.D., and Seymour Ochsner, M.D.
New Orleans, La.

PNEUMONECTOMY for pulmonary disease is now so widely used that it is difficult to realize that the first successful resection was not accomplished until April, 1933.[3] The great strides that have been made in thoracic surgery since then have occurred during the experience of many active surgeons. The first patient on whom one of us (A. O.) performed a pneumonectomy was seen recently, on the twenty-first anniversary of his operation. The rarity of pulmonary leiomyosarcomas and the long survival after pneumonectomy prompted us to report this case.

REPORT OF CASE

A white man, aged 19 years, was admitted to Touro Infirmary, March 10, 1936, complaining of spitting blood and pain in the left side of the chest. In the spring of 1935, he had consulted a physician because of cough and thoracic discomfort, but no abnormalities were discovered. By July, 1935, weakness and shortness of breath had developed. By October the cough had become productive and the sputum contained streaks of blood. On Nov. 12, 1935, the patient was examined at Sanatorium, Mississippi, and told that he had pleurisy, which could be tuberculous, although no tubercle bacilli were detected in the sputum. During late December, he had a severe attack of hemoptysis and spit blood for 3 days.

On Jan. 24, 1936, he returned to Sanatorium, Mississippi, for another examination, which showed extensive atelectasis of the left lung. Dyspnea gradually increased and episodes of hemoptysis recurred. The patient had constant pain in the chest, became anorexic, and lost 10 pounds in weight.

When admitted to Touro Infirmary, the patient was pallid and undernourished. The temperature was 99.6° F. and respirations, 20. Absent breath sounds and dullness to percussion on the left together with complete opacity of the left hemithorax and shift of the heart and mediastinum to the left, demonstrated in roentgenograms, indicated complete atelectasis of the left lung.

On April 2, 1936, a soft vascular tumor completely occluding the left main-stem bronchus was seen bronchoscopically. The tumor bled freely when tissue was obtained for biopsy. The histologic interpretation of this tissue was fibrosarcoma or leiomyosarcoma. On April 13, 1936, artificial pneumothorax was induced and the left pleural cavity was half filled with air.

Left pneumonectomy was performed on April 15, 1936. About 500 c.c. of serous fluid was found in the left pleural cavity. The left lung was firm and airless, and many nodes were palpable at the left hilum. Vessels at the hilum were individually ligated. The bronchus was doubly clamped and divided. The pathologist who examined the lung immediately after its removal reported that there was no bronchial tumor within the specimen. The bronchoscopist, who was present at the operation, stated that the tumor was just beyond the tracheal bifurcation. Thereupon the remaining segment of the left main bronchus was excised completely, including a portion of the trachea, which was closed obliquely. In this removed portion of the bronchus there was a tumor which largely filled the bronchus.

From the Departments of Surgery and Radiology, Ochsner Clinic, New Orleans, La.
Received for publication Oct. 28, 1957.

SINGLE DOSAG[illegible] [illegible]ATION TO THE LUNG AND POSTERIOR MED[illegible] THROUGH THE OPEN CHEST

I. RESULTS [illegible] DOSAGE BETWEEN 6,000 AND 15,000 R

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IN 1912, Torek [illegible] the first successful esophagectomy for carcinoma of the esophagus [illegible] years later, Graham[3] was able to do a pneumonectomy for carcinoma of the lung with long-term survival. Even with subsequent advances in the treatment of these two diseases the prognoses are not good. The resectability rate in cancer of the lung is variously given as 29 per cent[4] and 41 per cent[2] and the 5-year survival figure as 5.3 per cent.[4] Of the patients having a resection 31.7 per cent[2] were alive at the end of 5 years. The 5-year survival rate for carcinoma of the esophagus varies between 0 and 5 per cent.[5] Another series is more encouraging.[7] Here 6 per cent of the patients operated upon for midesophageal carcinomas and 15 per cent of those operated upon for lesions at the lower end of the esophagus were alive after 5 years. If the tumor was localized or metastatic only to regional lymph nodes, the rates, respectively, became 23 and 32 per cent. These percentages fall sharply if based on the total number of patients in the series. Both conventional x-ray therapy and chemotherapy have had trials but at the present time they offer only palliation in selected cases in most instances.

The dismal situation of low resectability and cure rate for carcinoma of the lung prompted Chesney and Daughtry[1] to radiate the lung through the open chest at the time of thoracotomy in some of the patients that they deemed nonresectable. As yet, their experience is meager and their approach has necessarily been cautious.

PURPOSE AND METHOD

Information on single dose x-ray radiation administered directly to the lung or mediastinum without the interposition of the chest wall is lacking. A long-term study was undertaken to investigate this modality of therapy because of possible direct clinical application. Patients with nonresectable tumors of the lung, esophagus, or posterior mediastinum might be candidates for this type of treatment. Patients whose tumors had been resected, but in whom there were positive regional lymph nodes, might also benefit from such therapy. The purpose of the investigation, then, was to establish the maximum safe

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Pulmonary resection for sarcoma of the lung was apparently first achieved by Lilienthal[5] in January, 1933, but the patient died on the fourth postoperative day and necropsy proved the pulmonary lesion to be a metastatic mass. In July, 1933, Archibald[1] successfully performed a pneumonectomy on a patient with a sarcoma of the upper lobe and, in October, 1933, Rienhoff[8] removed the left lung of a 3-year-old child who had a fibrosarcoma. So far as we are aware, our patient represents the first case of primary leiomyosarcoma of the lung successfully treated by pneumonectomy. In a recent review of primary pulmonary sarcomas,[7] we discovered reports of 15 cases of leiomyosarcoma of which 7 were resectable.

This case exemplifies several cardinal points in the diagnosis and management of pulmonary tumors.

1. Persistent hemoptysis calls for complete clinical, roentgenologic, and bronchoscopic examinations.
2. Extensive atelectasis may result from small tumors and even complete atelectasis of a lung does not mean that resection may not be successful.
3. A neoplasm may involve the bronchus quite close to the trachea. Successful excision may require complete removal of the bronchus and, at times, even a portion of the trachea.
4. Surgical removal of pulmonary sarcomas is desirable and, as in our case, may result in a long and useful life.

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subdivided according to the amount of radiation they received Group II comprised those animals that received radiation directly to the mediastinum with the lung protected Statistically the 2 groups did not lend themselves for comparison or was such an analysis of the subgroups possible Certain observations were, however, valid Tables I, II, and III record the gross post mortem findings of the first group and Tables IV and V do the same in those dogs receiving radiation directly to the mediastinum No effort was made to estimate accurately the body surface of the animals but the tables do record the body weights Survival time, complications, and clinical course did not correlate with the body weights

TABLE I*

DOG	WEIGHT (KG)	HGB	W B C	SURVIVAL IN DAYS	PLEURAL FLUID	REACTION TO LUNG	PNEUMONITIS	MEDIASTINAL REACTION	ESOPHAGITIS
2	11 8	--	--	10	+	++++	0	+++	++
4	14 0	--	--	42	++++	+++	++	0	0
7	13 6	10 2	11 200	103	†	+++	++	?	?
8‡	18 1		-	10	++	+++	+++	+	+
11§	18 1	9 2	10 000	60	0	++++	++++	+++	+
16	15 4	8 6	7 450	32	0	+++	+++	+++	+++

*Group I—Dogs receiving a single exposure of 12 000 r in air to the exposed lung surface and a depth dose of 7,800 r (7 254 RAD) to the esophagus

†250 c c fresh blood

‡This dog received 15 000 r

§Heart showed reaction to radiation

TABLE II*

DOG	WEIGHT (KG)	HGB	W B C	SURVIVAL IN DAYS	PLEURAL FLUID	REACTION TO LUNG	PNEUMONITIS	MEDIASTINAL REACTION	ESOPHAGITIS
9	11 8	10 6	11,300	27	++++	+++	0	+++	+++
10	22 7	9 9	15,300	25	++	++++	+++	0	0
12	15 9	9 9	18,000	16	++++	++++	0	+++	0
13	15 9	9 0	7,300	26	++++	+++	++	++	+
14	15 9	13 9	10,750	27	+	+++	+++	+++	+++
15	15 9	11 0	13,700	23	+	++++	++	++	+

*Group I—Dogs receiving a single exposure of 10 000 r in air to the exposed lung surface and a depth dose of 6 500 r (6 025 RAD) to the esophagus

TABLE III*

DOG	WEIGHT (KG)	HGB	W B C	SURVIVAL IN DAYS	PLEURAL FLUID	REACTION TO LUNG	PNEUMONITIS	MEDIASTINAL REACTION	ESOPHAGITIS
1	17 7	--	--	13	†	0	++	+	+
3‡	11 3		--	240+					
5	25 0	-	--	13	§	+++	+++	0	0

*Group I—Dogs receiving a single exposure of 8 000 r in air to exposed lung surface and a depth dose of 5 200 r (4 836 RAD) to the esophagus

†Pleural fistula wound not well protected

‡Received 6 000 r dog still living

§Empyema

The hemoglobin in milligrams per cent, and the white blood cells per cubic millimeter were recorded These determinations were done at the time of thoracotomy and then at weekly intervals Those in the tables are the initial values As has been stated, the hemoglobin and white count reflected the dose of x-ray radiation that may be given by this method, to estimate if such doses were cancerocidal, to determine if they produced less systemic effect than conventional forms of therapy, and to determine what complications can be expected. It was also the aim of the study to search for means of eliminating or treating complications. Finally, assuming that a suitable technique was developed and that doses were cancerocidal, information about pulmonary function before and after treatment must be gathered.

Mongrel dogs weighing between 9 and 27 Kg. were used. The state of health of the animals varied considerably but there was a coarse correlation between the hemoglobin and white cell count with the clinical condition of the dog. In south Florida, many of the mongrel dogs are underweight, many have heart worms, and most are infested with intestinal parasites. Under intravenous veterinary Nembutal anesthesia, the dogs' tracheas were intubated and connected to a respirator. A thoracotomy in the right fourth interspace was performed under sterile conditions. A circular cone made of sterile sheet lead was inserted into the posterior aspect of the wound in such a way that the tissues surrounding the portal of radiation entry were completely protected by at least 2 mm. of lead. The entry portal measured 28.26 sq. cm. In one group of dogs, the radiation was directed through the lung toward the posterior mediastinum. In this group, care was taken to see that there were no atelectatic areas during radiation. In the second group, the lung on the side that was operated upon was packed away from the entry portal, thus allowing the radiation to pass directly to the mediastinum without going through the lung. Following administration of radiation, the chest was closed in layers using continuous sutures of silk. Some of the dogs received antibiotics but in this series it neither affected the survival time nor the complication rate. Postoperatively, the dogs were fed standard kennel rations and were observed for symptoms. Hemograms and chest films of the animals were obtained at intervals. At the time of death, post-mortem examinations were done on all dogs.

The dose of radiation delivered varied but the factors of distance and quality remained constant. The radiation used was generated by a self-rectified therapy unit operated at 250 PKV, 30 Ma., at surface-target distance of 25 cm. and with an output of 1,200 r per minute in air at the surface of entry. The quality of the entry radiation was 4.0 mm. aluminum half-value layer. In vivo determinations and measurements made of the isolated respiring lungs of a dog showed a depth dose in the mediastinum at the level of the esophagus to average 65 per cent of the entrance surface dose when the radiation passed through the lung. The periphery of the opposite lung received 40 per cent. The absorbed dosages are expressed in RADs (radiation absorbed dose) and in tissue roentgens at the depth in question.

RESULTS

The dogs have been divided into 2 groups. Those receiving radiation through the lung have been designated as Group I and have been further

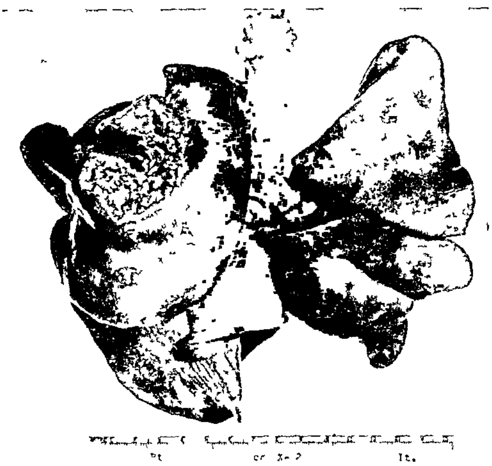

Fig 1—A photograph of the lungs of Dog 12 (Table II) shows necrosis of the radiated lung with excavation The left lung and the remainder of the right lung display only minimal changes This dog received 10,000 r directly to the lung 16 days before death

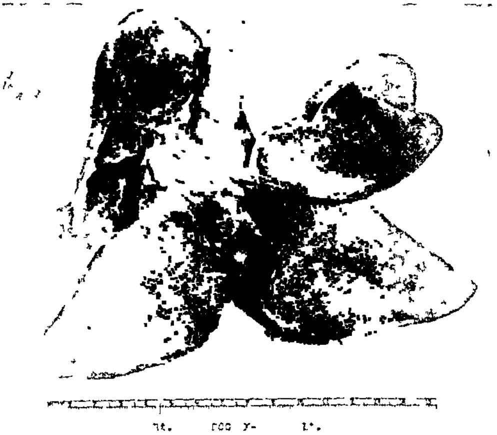

Fig 2—A photograph of the lungs of Dog 4 (Table I) shows radiation reaction less intense than that in Fig 1 This animal received 12,000 r directly to the lung 42 days before death

TABLE IV*

DOG	DOSAGE OF RADIATION	WEIGHT (KG.)	HGB.	W.B.C.	SURVIVAL IN DAYS	PLEURAL FLUID	REACTION TO LUNG	PNEUMONITIS	MEDIASTINAL REACTION	ESOPHAGITIS
18	15,000 r	13.6	8.4	9,000	3	++++	0	+	++	+
19	15,000 r	18.6	9.6	22,700	14	+	0	++	+++	+++
20	15,000 r	18.6	8.4	16,000	12	0	0	+++	+++	+++
21	15,000 r	11.3	6.6	7,900	4	+++	0	+++	0	0
22	12,000 r	9.5	7.9	6,600	12	++	0	0	+++	0
23	12,000 r	10.4	9.7	22,800	20	0	0	+++	+++	+++

**Group II*—Dogs receiving a single exposure of 12,000 to 15,000 r (11,160 to 13,950 RAD) to the depth of the esophagus with the lung shielded.

TABLE V*

DOG	DOSAGE OF RADIATION	WEIGHT (KG.)	HGB.	W.B.C.	SURVIVAL IN DAYS	PLEURAL FLUID	REACTION TO LUNG	PNEUMONITIS	MEDIASTINAL REACTION	ESOPHAGITIS
24	8,000 r	--	9.8	14,500	6	+	0	++	+	0
25	8,000 r	9.0	10.2	6,200	64	+	0	++++	?	?
26†	8,000 r	27.2	19.1	11,600	100+‡					
38	6,000 r	15.0	--	--	2	0	0	++	0	0

**Group II*—Dogs receiving a single exposure of 6,000 to 8,000 r (5,680 to 7,440 RAD) to the depth of the esophagus with the lung shielded.

†Greyhound.

‡Still living.

general health of the animal with some accuracy. During the postradiation (and postoperative) course there was no recognizable change in the hemoglobin for all the dogs or for any group or subgroup. Generally speaking, if the animal was malnourished and had a low hemoglobin it rose as a result of the routine kennel ration that he received. If the animal developed esophagitis and stopped eating, the hemoglobin fell. The radiation had no effect on the hemoglobin in the animals recorded in the tables. Similarly the white blood cell counts that were listed were the initial values. Here too there was no discernible pattern. If the dog developed pneumonitis from the radiation with secondary infection, the white count rose; otherwise, it remained unchanged. In no subject did a leukopenia develop.

The x-ray radiation dose within the range administered did not affect the survival time. Thus, the dogs receiving 6,000 r did not routinely live longer than those receiving 15,000 r. Because of the small size of the groups this does not have statistical significance. Similarly no difference in survival time existed if the dog received the radiation directly to the mediastinum or through the lung. Two dogs survived. One was a greyhound in excellent physical condition at the time of thoracotomy. At post-mortem examination many of the dogs had significant pleural effusions. Those that were graded four plus had between 700 and 900 ml. of fluid present in the pleural spaces. By far the greater amount of fluid was present on the right, which was the side of thoracotomy, but often fluid was also present on the left. This was understandable when it was realized that the left lung received 40 per cent of the radiation administered to the surface of the right. As a general rule those dogs that had

"Pneumonitis" in the tables referred to changes in the left lung and in areas of the right lung that were not directly within the field of radiation. Pneumonitis was used here to mean pneumonia, atelectasis, or pulmonary edema. Usually all three were present in varying degrees. Chest films showed pneumonitis within the first 7 days but these x-ray findings did not persist even if considerable pathologic change was found at autopsy.

Mediastinal reaction consisted of edema, induration and later fibrosis of the posterior mediastinum. Changes here paralleled the esophagitis that developed but occurred earlier. The esophagitis was always more intense in the Group II dogs and in some instances the trachea and bronchi were the site of severe reactions. Fig. 3 shows only minimal changes in the esophagus of a dog receiving radiation through the lung. Fig. 4 demonstrates mucosal sloughing in a dog receiving radiation directly to the mediastinum. Esophageal perforation or fistula did not occur. Clinically the animals that developed esophagitis stopped eating about 10 days following radiation. Many of them developed a cough that was due to an associated tracheitis, bronchitis, or both. Some dogs then improved only to stop eating again a day or 2 before death.

The clinical course of the 2 groups was slightly different. All dogs recovered well from thoracotomy and radiation. On the first postoperative day they exhibited little or no discomfort, walked about and ate and drank well. The dogs in Group I for the most part did well until one or 2 days before death. Those in Group II also did well initially but about 10 days postoperatively they developed signs of esophagitis and tracheitis as manifested by cough and failure to eat. Often there would be transient improvement only to be followed by a recurrence of symptoms which shortly preceded death.

DISCUSSION

Single dosage x-ray radiation to the lung and posterior mediastinum through the open chest was a simple procedure. The radiation was immediately completed rather than prolonged. Systemic manifestations, such as radiation sickness, were absent with this method of administration. Anemia and leukopenia did not develop. With the protection used, there was no delay in wound healing and no skin damage. All of these animals received much larger than calculated cancerocidal dosages of x-ray radiation through a relatively large port. With the dosage used, it was not surprising that all animals (except 2) died. Death was due to direct destruction of tissue or a complication of tissue damage. Thus mortality was from pleural effusion, pneumonitis, or esophagitis in almost all instances. Possibly some of the dogs would have survived longer had their complications been treated. Because of the much higher than cancerocidal doses used in this series, no conclusions regarding clinical application can be made. Further laboratory work with lower but still cancerocidal dosages is indicated. A different technique of radiation administration with or without the addition of a chemotherapeutic agent bears investigation.

radiation through the lung (Group I) had more pleural effusion at autopsy than did those that received it directly to the mediastinum (Group II). Almost all of the animals developed some pleural effusion within the first week following radiation as indicated by serial x-ray studies. In addition to a massive pleural effusion, Dog 4 had a pericardial effusion large enough to cause death from tamponade. This occurred early in the series and all subsequent dogs had windows cut in the pericardium.

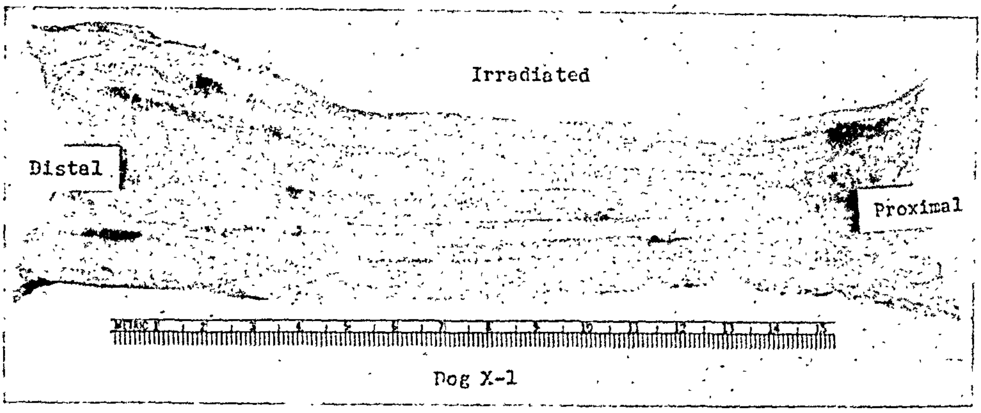

Fig. 3.—A photograph of the esophagus of Dog 1 (Table III) shows mild erythema in the radiated area. Radiation was through the lung.

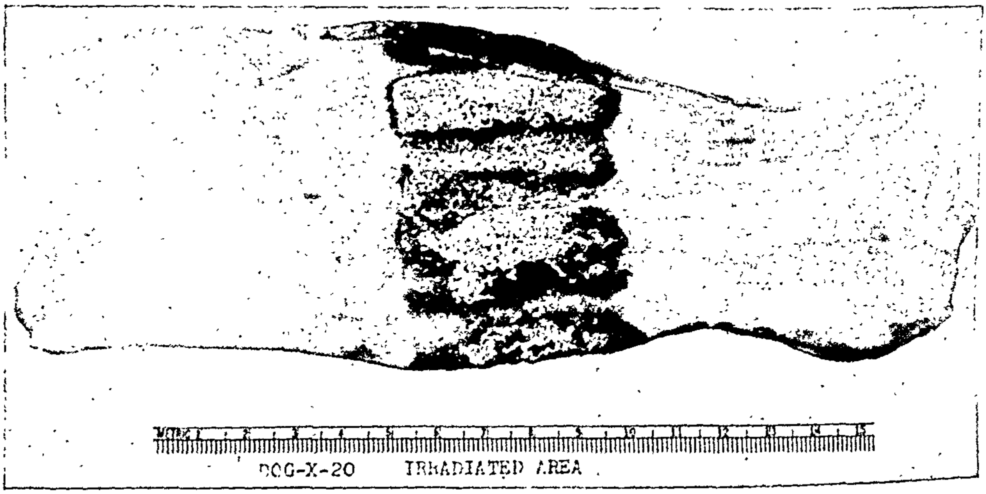

Fig. 4.—A photograph of the esophagus of Dog 20 (Table IV) shows an intense reaction with sloughing of the mucosa in the radiated area.

The reaction of the directly radiated lung in Group I varied. Pulmonary reaction in the dogs receiving the higher dosage was not uniformly greater than in those receiving 6,000 r. Fig. 1 shows the typical gross appearance of the lung in Group I dogs that were graded four plus. Fig. 2 shows a less intense reaction. On serial x-ray studies the lung reaction appeared as a circumscribed density within the first week and persisted on subsequent films until the animal finally died.

HYDATID CYST OF THE LUNG WITH BRONCHOGRAPHIC EVALUATION OF TREATMENT BY INTERNAL SUTURE OF THE PERICYST

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HYDATID disease (echinococcosis) is a common parasitic infection in certain sheep- and cattle-raising areas. South America, Australia, New Zealand, North and South Africa, and the Middle East are particularly affected. Cases from these endemic regions will appear elsewhere with greater frequency as world travel increases and it is also possible for other endemic foci to occur wherever conditions are favorable to the parasite. Thus, it is likely that the clinical features and treatment of various aspects of this disease will be of more general interest in the future. For this reason we are presenting our experience with cases of hydatid cyst of the lung in the American University Hospital during the past 10 years (July 1, 1947 to June 30, 1957).

PARASITOLOGY

Before dealing with clinical problems, a brief review of the life history of the responsible parasite may be helpful. Hydatid cysts are the larval stage of the minute tapeworm, *Echinococcus granulosus*, which infests the intestine of dogs and dog-like animals (the definitive hosts). The worm, which is only 2 to 8 mm. in length, consists of a scolex and neck followed by three or four successively larger segments. The terminal segment, or proglottid, contains eggs which are passed in the dog's stools to contaminate the food or water consumed by certain herbivorous mammals such as cattle, sheep, and human beings (the intermediate hosts). Human infection, especially in the case of children, may also result from such intimate association as playing or living with dogs. When the eggs are ingested by an intermediate host, they hatch in the upper gastrointestinal tract to release a larval embryo which penetrates the bowel wall. The embryo then enters the portal circulation or, less frequently, the lymphatics or systemic veins. Most of the embryos will be filtered out in the liver with the lungs being the second most commonly affected organ. Embryos may occasionally escape these two filters and lodge in other parts of the body. Multiple organ involvement is not uncommon.

After the larval embryo has come to rest, and if it is not destroyed by the humoral and tissue reactions of the host, it develops into an echinococcus cyst. This lesion is appropriately called a hydatid or a hydatid cyst since the

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SUMMARY AND CONCLUSIONS

Through a thoracotomy, one group of dogs received single rather high doses of x-ray radiation directly to the lung while a second group received it to the posterior mediastinum. In the first group, death was the result of pleural effusion or pneumonitis. In the second group, esophagitis with or without tracheitis caused death. Further investigation is warranted using lower but still cancerocidal doses of radiation.

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small, thin walled blisters or pedunculated cysts on its inner surface These are known as *brood capsules* and in them are formed tiny scolices which may number several million in a large cyst Detached and free floating brood capsules and scolices make up a fine sediment called *hydatid sand* in the cyst fluid

If the germinal membrane itself either invaginates or evaginates, a portion of it may be pinched off to form a *daughter cyst* which has the characteristic laminated membrane, hydatid fluid, brood capsules, and scolices These daughter cysts may also develop from brood capsules or scolices The daughters are usually inside but may be outside the mother cyst membrane the continuity of which is broken by the irritant or aging process which has stimulated the daughter cyst formation[2] Daughter cysts are fairly common in hydatids of the liver but are infrequent in the lung In our 77 pulmonary hydatid cysts, only 3 had daughter cysts

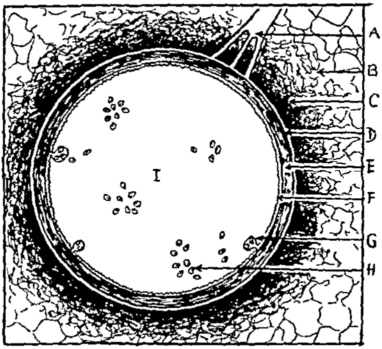


Fig 1—Schematic representation of a simple primary hydatid cyst of the lung *A* Bronchi *B* Lung parenchyma *C* Pericyst (or adventitia) *D* Potential space between pericyst and parasite *E* Laminated layer of parasite membrane (ectocyst) *F* Germinal layer of parasite membrane (endocyst) *G* Brood capsule containing scolices *H* Free floating scolices *I* Hydatid fluid

Both cyst fluid and scolices are a particular menace Due to the fact that the host develops more or less antibodies in response to the parasite, the leakage of fluid into the tissues after spontaneous or surgical rupture of the cyst wall may produce any of the whole gamut of immunologic reactions from urticaria to fatal anaphylactic shock The scolices, which readily escape as the fluid leaks, will implant themselves and produce *secondary hydatid cysts* which are so called to distinguish them from the *primary hydatid cysts* that arise directly from ingested ova Because of the risks of anaphylaxis and implantation, spillage of hydatid fluid must be carefully avoided during surgical treatment

The hydatid is not attached in any way to the host but receives all its nourishment by osmosis through the cyst membrane As the cyst grows, the host tissues are compressed to form an encircling, fibrotic sh term "hydatid" is derived from a Greek root meaning "watery vesicle." The growth of the cyst is slow and insidious, the rapidity depending on the environment. At the end of 6 months it will usually measure only 1 to 2 cm. in diameter but eventually it may reach a huge size and contain several liters or more of fluid. Pulmonary cysts larger than 20 cm. are not common but hydatids filling an entire hemithorax are occasionally seen. Barrett and Thomas[1] have noted cysts to grow to 6 cm. diameter in the lung within a year. It is also frequently observed that a cyst may remain fairly constant in size for several years at a time.

Table I gives the organ distribution in the 176 patients with hydatid cyst seen at the American University Hospital during the past 10 years. The diagnosis in all cases was proved by operation or autopsy. The relative incidence of pulmonary cysts in our experience (32 per cent) is somewhat higher than that usually reported. For example, Dévé[4] noted liver cysts in 75 per cent of cases and pulmonary cysts in 10 per cent. Chandler[2] records a 60 to 75 per cent liver involvement as compared with a 20 per cent lung involvement. On the other hand, Craig, Faust, and Miller,[3] state that in Chile pulmonary hydatid is as common as hepatic hydatid. It is most probable that, instead of a varying predilection of the parasite, the composition of statistics from different institutions is influenced by extraneous factors.

TABLE I. ORGANS INVOLVED IN 176 CASES OF HYDATID CYST

CASE NO.	LIVER	LUNG	SPLEEN	KIDNEY	HEART	PERITONIUM	MISCELLANEOUS
A. Distribution of 164 Cases of Hydatid Cyst With Involvement of a Single Organ							
	84	59	3	3	1	3	11*
B. Distribution of 12 Cases of Hydatid Cyst With Multiple Organ Involvement							
1	x	x					
2	x	x					
3	x		x				
4	x		x				
5	x			x			
6	x			x		x†	
7	x					x†	
8	x					x†	
9	x						
10		x	x				
11		x†			x		
12			x	x			

*Soft tissues 4, orbit 2, spine 2, brain 2, thyroid 1.

†These were unquestionably secondary cysts due to rupture of a primary cyst in the other organ listed.

An understanding of the anatomy of the hydatid as shown in Fig. 1 is important. The parasite itself is a fluid-filled cyst, the wall of which is composed of an opaque, white, friable membrane that is paper-thin during early development and eventually reaches only a few millimeters in thickness. The cyst wall is actually composed of two layers—an inner germinal and an outer acellular layer which is laid down in concentric laminae by the internal germinating surface as a protective covering around itself. The germinal membrane secretes a water-clear hydatid fluid into the cyst cavity which is thereby tensely distended to a globular shape. The germinal surface, if it is fertile, produces

Total patients: 62 of which 4 were in the age range of 0-10 yr., 13, 11-20 yr.; 16, 21-30 yr.; 11, 31-40 yr.; 12, 41-50 yr.; 4, 51-60 yr.; 2, 61-70 yr. The youngest patient was 5 and the oldest 65 years old.

Sex distribution: There were 44 males and 18 females.

Incidence of complicated cyst: 33 of the 62 patients had complicated cysts.

Multiple cysts: 11 patients had multiple cysts. Bilateral cysts occurred in 5 patients. The total cysts in 62 patients was 77.

Daughter cysts: Three cysts (all complicated) contained daughter cysts

Location of 77 cysts: There were 45 (58 per cent) on the right side: upper lobe, 14; middle lobe, 11; lower lobe, 20 On the left side, there were 32 (42 per cent): upper lobe, 13; lower lobe, 19.

Size of 73 cysts recorded: 1-5 cm., 17; 6-10 cm., 48, 11-15 cm., 7; 16-20 cm , 1.

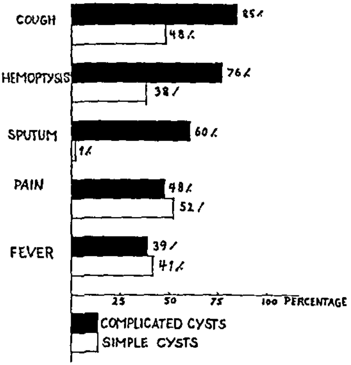


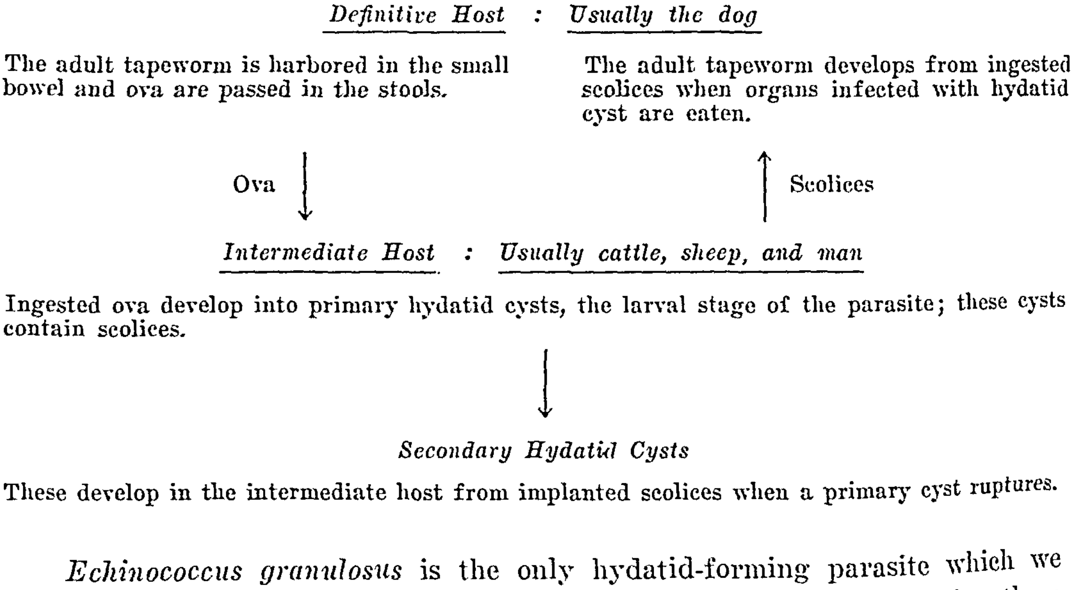
Fig 3.—The incidence of various symptoms in 62 patients with pulmonary hydatid cyst

Symptoms.—Simple cysts cause no complaints until pressure symptoms or pericystic pneumonitis in compressed or obstructed lung tissue occur. Seven of our patients had the diagnosis made on chest radiography or fluoroscopy done for another purpose. The incidence of asymptomatic cysts discovered in this manner will doubtless increase in the future.

Complicated cysts are the result of rupture and infection. Their symptoms vary over quite a wide range. The patient may cough up clear fluid or fragments of cyst membrane. A large membrane in the tracheobronchial tree may cause localized obstruction or even death from asphyxia. In rare instances, the cyst may rupture into the pleural space to produce pyopneumothorax, and pleural implantation of secondary cysts may follow. Widespread pneumonitis occasionally gives the picture of acute pneumonia or pulmonary abscess More or less purulent sputum is common. On the whole, however, the which in the larger pulmonary cysts may be a half centimeter or more in thickness. This zone is known as the *pericyst* to distinguish it from the parasite itself. The expansion of the hydatid almost invariably erodes one or more small bronchi which open into the potential space between pericyst and the parasite. Fluid leaking from the hydatid may therefore enter the bronchial tree, and bacteria from the respiratory passages have access to the pericyst cavity. When pulmonary cysts rupture spontaneously, as they frequently do, both pericyst and hydatid promptly become infected. This results in death of the parasite and its scolices. An infected hydatid will no longer implant or cause anaphylactic reactions. Viable intact hydatids (whether primary or secondary) are termed *simple* cysts. Ruptured or infected hydatids are considered *complicated* cysts.

The life cycle of this sinister parasite is completed when the cyst-containing viscera of intermediate hosts, such as sheep and cattle, are eaten by dogs. The ingested scolices develop into adult tapeworms in the dog's intestines (Fig. 2).

Fig. 2.—Life cycle of the tapeworm, *Echinococcus granulosus.*



Definitive Host : *Usually the dog*

The adult tapeworm is harbored in the small bowel and ova are passed in the stools.

The adult tapeworm develops from ingested scolices when organs infected with hydatid cyst are eaten.

Ova

Scolices

Intermediate Host : *Usually cattle, sheep, and man*

Ingested ova develop into primary hydatid cysts, the larval stage of the parasite; these cysts contain scolices.

Secondary Hydatid Cysts

These develop in the intermediate host from implanted scolices when a primary cyst ruptures.

Echinococcus granulosus is the only hydatid-forming parasite which we have encountered in the Middle East. In Europe, Siberia, and Alaska, there is seen an alveolar form of echinococcosis characterized by the formation of multiple thin-walled vesicles which infiltrate the surrounding tissues. It has been suggested by Rausch and Schiller[7] that this alveolar hydatid disease is caused by the larval stage of *Echinococcus sibiricensis* which is a tapeworm morphologically almost identical with *E. granulosus.*

PULMONARY CYSTS AT THE AMERICAN UNIVERSITY HOSPITAL

General Data.—During the past 10 years, 62 patients with proved primary hydatid cyst of the lung have been treated. A few of the characteristics of this series follow.

present, is a narrow crescent of decreased density around the margin of the mass This is due to air between the hydatid and pericyst (Fig 4, *A*) Tomography is useful in demonstrating the crescent more clearly When part of the fluid has escaped from the parasite, the crescent may become quite prominent If air also enters the hydatid cyst there may be two cavities, one above the other (Fig 4, *B*) After complete rupture of the parasite with collapse of the membrane, an abscess cavity results An air fluid level is frequently present, the surface of which may be wavy or irregularly scalloped due to the crumpled, retained membrane (Fig 4, *C*) This "water lily" sign may also be produced by necrotic debris in an abscess cavity of other origin

Bronchography is not particularly helpful

Bronchoscopy—In complicated cysts, the bronchoscopist may establish the diagnosis by removing a portion of membrane which has prolapsed into an accessible bronchus In rare instances, the entire membrane can be teased out and healing of the pericyst, without surgical intervention, becomes a possibility Bronchial washings should be searched for membrane shreds if no gross fragments are obtainable

Blood Tests—The immediate Casoni skin sensitivity test and the Weinberg complement fixation test are based on immunologic reactions If positive, then hydatid infection has probably occurred, if negative, the disease is not ruled out False positives occur occasionally, especially with the Weinberg test We use these tests regularly to obtain supporting evidence but the diagnosis cannot be based upon them in endemic areas In parts of the world where echinococcosis is rare, a positive Casoni or Weinberg would be more useful

Susman,[8] quoting the experience of Dew and others, reports the immediate Casoni reaction to be positive in 75 per cent of simple and in 92 per cent of complicated cysts The Weinberg test is less sensitive and is said by the same author to be positive in 50 to 60 per cent of cases Waddle[9] has reviewed 478 cases of pulmonary hydatid which occurred in Australia and New Zealand over a 30 year period In proved cases of hydatid disease, the Casoni was po[illegible]tive in 57 per cent, the Weinberg was positive in 55 per cent, and one or b[illegible] were positive in about 75 per cent Our limited experience with these two [illegible]ts in pulmonary echinococcosis in 49 patients is summarized as follows

Casoni Neg, Weinberg Neg	11 (22%)
Casoni Pos, Weinberg Neg	13 (27%)
Casoni Neg, Weinberg Pos	11 (22%)
Casoni Pos, Weinberg Pos	14 (29%)

One or both of the tests were positive in 78 [illegible] There [illegible] ference in the percentage of positive tests in [illegible] and compl[illegible] our cases

Eosinophil count was done in 61 cases [illegible] following [illegible]

Normal	0 4%	[illegible] cases	[illegible]
Abnormal	5- 9% 10 15% >16%	[illegible] 17 Cases	[illegible]

patient with a complicated cyst is not as sick as one would expect from the appearance disclosed by x-ray studies and he may even have minimal or no complaints for long periods.

The commonest symptoms of both simple and complicated cysts are cough, purulent sputum, hemoptysis, pain, and fever. The incidence of these complaints in our patients is shown in Fig. 3. It is important to note the high incidence of hemoptysis. Streaking of sputum with bright blood intermittently is most frequent but hemoptysis up to 100 ml. of blood has occurred rarely in this series. Variations between these two extremes are not unusual. The chest pain is characteristically mild or moderate and localized to the region of the chest wall near the cyst. It ranges from pleuritic to an ill-defined dull ache. The fever occurs either in acute exacerbations with symptoms suggestive of respiratory infection or as a low-grade chronic pyrexia. It is common for the symptoms of hydatid cyst to begin with an episode suggestive of pneumonia. It is surprising to find that fever has been so frequent in simple cysts; it should be added that the duration and intensity of fever is characteristically greater in the complicated cases. The duration of symptoms prior to admission in 62 patients follows.

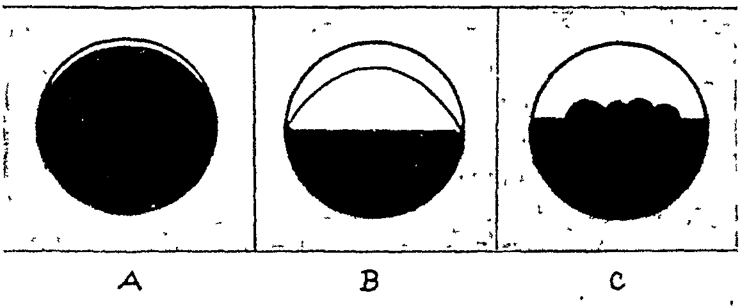


Fig. 4.—Diagrammatic representation of some typical x-ray findings (after Dévé). *A*, A tiny crescent of air may sometimes be seen between parasite and pericyst. When present it is practically pathognomonic but many cysts do not show this sign. *B*, A double space may result when air enters a ruptured hydatid. *C*, A "water-lily" sign is frequently seen in ruptured hydatid cyst The collapsed hydatid membrane scallops the air-fluid level. Taking films with the patient in lateral decubitus or other positions may aid in visualizing the membrane.

	Simple Cysts	*Complicated Cysts*
< 6 months	16	13
7-12 months	3	9
> 1 year	4	10
No symptoms	6	1

Radiology.—Chest radiography is essential to diagnosis unless the patient actually coughs up a membrane. Dévé[4] has summarized the roentgenographic signs in a series of diagrams of which Fig. 4 is a modification. Where echinococcosis is endemic, clinicians are generally quite familiar with the radiologic appearance of the lesion and most cases are correctly diagnosed preoperatively. The simple hydatid appears as a sharply defined oval or spherical shadow of homogeneous density in the lung field and is easily confused with tuberculoma or neoplasm. The rounded shadow of a cyst may become oval on deep inspiration (Escudero-Nemerow sign). An even more suggestive finding, when treatment in most cases. Our results, to be discussed below, confirm this opinion. There are 3 phases of this operation deserving comment: (1) the exposure, (2) removal of the parasite, and (3) closure of the pericyst.

1. *Exposure:* This is obtained by a wide thoracotomy using an anterior, lateral, or posterior (face-down) approach depending on the location of the cyst. All adhesions are divided so that the lung is completely mobilized. This permits positioning of the cyst in the middle of the operative field with careful walling-off of pleural space and wound by moist gauze. Also important, the lung surrounding the cyst relaxes easily to permit secure closure of the pericyst and then expands freely to obliterate the space previously occupied in the thorax by the mass of the cyst.

2. *Removal of the parasite:*

a. *Complicated cysts:* If the membrane is collapsed and infected, then the scolices will not be viable and no special precautions to prevent their implantation are required. The parasite is easily managed by opening the avascular peripheral dome of the pericyst, extracting the macerated cyst membrane with sponge forceps and swabbing out the accumulated pus and debris with dry gauze. The interior of the pericyst is only mechanically cleaned and may then be closed as described below.

b. *Simple cysts:* These must be handled with care in order to avoid spillage of hydatid fluid and scolices with the danger of immediate anaphylactic shock and of delayed secondary cyst formation. At this stage of the operation, a quiet operative field is most desirable and the anesthetist is requested to minimize the respiratory motions, using muscle relaxants if necessary. The pericyst is carefully incised down to the glistening white membrane of the hydatid which tends to bulge into the pericyst incision, especially if the anesthetist applies too much positive pressure. The opening in the pericyst is cautiously lengthened for several centimeters and the margins are held up and apart by Allis forceps. Protective gauze packing is moved up to the edge of the pericyst incision. A 22-gauge needle connected to a 3-way stopcock and syringe is inserted into the cyst while a fenestrated suction tip is held near the puncture site to catch any leakage of fluid. Usually from 10 to 30 ml. of hydatid fluid are aspirated so that the cyst membrane relaxes slightly without collapsing. This fluid is evacuated into a basin through a length of rubber tubing attached to the side-arm of the 3-way stopcock. Another syringe containing formaldehyde, U.S.P. (approx. 35 per cent formaldehyde) is attached to the 3-way stopcock. The formaldehyde is slowly injected through the needle which has remained in place held steadily by an assistant. The volume of the cyst is estimated and the amount of formaldehyde is used which will result in a 1 per cent solution within the cyst. This concentration should kill the scolices in 5 minutes.[4] With the fine needle still in place to avoid leakage, 5 minutes are allowed to pass at the end of which time a second and larger needle (e.g., 15-gauge, 12 cm. length) is introduced carefully into the cyst the contents of which are aspirated using a 50 ml. syringe and 3-way stopcock with rubber tubing attached to the side-arm. Again the fenestrated suction tip is held near the site of puncture in the

Since intestinal parasites were very common among the patients of this series it is difficult to know the relation of eosinophilia to echinococcosis in these cases. The eosinophil count is not very helpful in differential diagnosis.

TREATMENT

Hydatid cysts of the lung should be removed when they are discovered if the patient's condition warrants. Simple, asymptomatic cysts are no exception since they will grow to troublesome proportions and there is the ever-present danger of spontaneous rupture. Furthermore, surgical treatment is safe and prospects for cure are excellent.

The various types of treatment carried out on our 62 cases are indicated in Table II. We have abandoned the method of marsupialization of the cyst to the chest wall followed by open drainage of the pericyst cavity since bronchocutaneous fistula and residual abscess are troublesome sequelae. We would now advise one of the following 3 techniques of treatment depending upon the circumstances of the case: (1) pulmonary resection, (2) dissection of the pericyst, or (3) removal of the hydatid membrane and internal suture of the pericyst.

TABLE II. TYPES OF TREATMENT IN 77 PULMONARY HYDATID CYSTS WHICH OCCURRED IN 62 PATIENTS

OPERATION	SIMPLE CYSTS	COMPLICATED CYSTS	TOTAL
Marsupialization and drainage	5	4	9
Lobectomy	1	10	11*
Dissection of pericyst	1		1
Evacuation of parasite and internal suture of pericyst	33	20	53
Nondescript	2	1	3
Total	42	35	77

*Actually only 10 lobectomies have been done since, on one occasion, 2 cysts were removed in the same lobe.

Pulmonary Resection.—It is rarely necessary to do a segmental, lobar, or more extensive resection for hydatid cysts because simpler measures suffice. We reserve resection for those cases in which sepsis in a complicated cyst has destroyed a lobe, produced neighboring bronchiectasis, or otherwise made it unwise to leave behind the pericyst and surrounding tissues. This, fortunately, is not common.

Dissection of the Pericyst.—In this technique, a dissection plane is developed just outside the pericyst which is completely enucleated with the intact parasite. The bronchi opening into the pericyst and the defect in the lung are then closed with absorbable sutures. We have had little experience with this procedure but consider it most suitable for peripherally located, simple cysts of 6 cm. or less in diameter.

Removal of the Hydatid Membrane and Internal Suture of the Pericyst.—We have found this to be the easiest, safest, and most satisfactory method of seems appropriate, until the pericyst is securely obliterated. The original incision into the pericyst is finally and tightly closed with running or Connell sutures so that there is no air leak. The chest is drained with a tube brought out through an interspace and placed on water-seal. Postoperative care includes antibiotics and the usual methods to maintain complete expansion of the lungs.

RESULTS

Treatment in our cases was carried out by a number of different surgeons. In the past 5 years most operations have been performed by members of the Resident Staff. There have been no operative deaths in the 62 patients who make up our total 10-year series.

The operation of hydatid evacuation and internal suture of the pericyst has been done all over the world for a number of years. Results appear to be satisfactory but detailed follow-up studies are still needed to complete the evaluation of this procedure. Since we have used internal suture more frequently than any other technique, we have concentrated on obtaining follow-up information on its efficacy and will now report our findings.

Forty-two patients with 53 cysts requiring a total of 46 thoracotomies have been treated by internal suture of the pericyst. There were 4 postoperative complications as follows: minor wound infection, 2; bronchocutaneous fistula, 1; and cerebral air embolism, 1.

The bronchocutaneous fistula consisted of a transient slight air leak through the site of a catheter which was sewn into the center of the obliterated pericyst cavity as a temporary drain. This was done in a few cases early in this series since it was feared that an undrained pericyst cavity might flare-up as an abscess in the postoperative period. This fear has proved to be unfounded. The patient with the fistula left the hospital on the fourteenth postoperative day and the fistula closed spontaneously shortly thereafter without treatment. The cerebral air embolism was the only significant complication encountered and a permanent left hemiparesis has resulted. This complication is likely unrelated to the operation of internal suture since air may have entered pulmonary veins which bled profusely when an unsuccessful and quickly abandoned attempt was made to dissect out the pericyst.

The duration of postoperative hospitalization required in 46 thoracotomies for internal suture is given in Fig. 5. The morbidity following this procedure has been satisfactorily low. It is necessary, however, to demonstrate that functional and anatomic results are equally satisfactory before confidence in the internal suture method can be finally established. It has been stated that the pericyst cavity will not heal completely and that a residual pocket of variable size will remain to suppurate later.[6] Also, most surgeons when doing an internal suture for the first time on a complicated cyst have doubtless shuddered at the prospect of wiping the pus out of an infected pericyst cavity and then sewing it up tightly. Unquestionably, we have to thank the antibiotics for making this relatively innocuous.

It has been possible for us to follow only 26 of the 42 patients on whom internal suture operations were done. Local conditions have made long-term follow-up difficult and many of our patients w cyst to pick up leakage, should it occur. As the hydatid cyst is decompressed it is possible with ease to open the pericyst widely without fear of rupturing the membrane. The pericyst margins are held up and apart by Allis forceps and the collapsing membrane is gently grasped with ring-type sponge forceps and lifted into an awaiting basin. Should leakage of fluid occur, the scolices will have been killed by the 1 per cent formaldehyde which is itself of insufficient strength to be seriously toxic to the tough pericyst lining. "Formolage," as this procedure is called, is not effective when daughter cysts are present. In these cases one must rely on aspiration alone. Daughter cysts are lifted out along with other contents of the pericyst. A large kitchen spoon or ladle may be needed in these circumstances. If there is spillage of hydatid fluid in the pericyst, it may be mopped out with 1 per cent formaldehyde solution followed by saline sponging. If this is done carefully no formaldehyde will get into the bronchial tree.

It is fortunately unlikely that scolices spilled in the pericyst cavity at operation will easily take on this leathery, avascular surface which inevitably becomes secondarily infected after internal suture. It is doubtless for this reason that Susman[8] and other Australian surgeons do not take the precaution of formolage when dealing with primary cysts of the lung but merely aspirate them and lift out the collapsed endocyst with surrounding tissues well protected. We have favored formolage as an additional precaution which is simple to carry out.

Barrett and Thomas[1] are even more assiduous in avoiding hydatid fluid spillage. In primary simple cysts of intermediate size, they advise the removal of the intact parasite. This is accomplished by a wide incision in the pericyst from which the hydatid is gently expressed by the expanding lung and the air in the pericyst when the anesthetist increases the positive pressure. These authors used in the method successfully in 17 cysts. In 4 others the cysts ruptured during the process of removal. We tried this procedure in 5 patients with cysts 10 cm. in diameter or smaller. In 2 cases the hydatid suddenly and unexpectedly ruptured while being eased out. In one of these patients, the spillage of hydatid fluid onto surrounding tissues coincided with an immediate drop of blood pressure which responded to vasopressors and was considered to be an evidence of anaphylactic shock. Although it is very satisfying to get out the parasite intact, it is our impression that the danger of uncontrolled spillage of hydatid fluid is great with this technique. For those not thoroughly experienced in judging the tensile strength of the cyst membrane, it is wiser to use the less difficult method of aspiration.

3. *Closure of the pericyst:* The objective is to obliterate the pericyst cavity as completely as possible with absorbable sutures. Chromic catgut (2-0) on an atraumatic needle is very satisfactory. The bronchial openings in the base of the cavity are individually closed if possible. In a large cyst it is rarely convenient to close them all and it is unnecessary as long as the walls of the cyst are well approximated. Working inside the pericyst cavity, deep bites in the pericyst wall are taken, purse-string and interrupted sutures being used, as

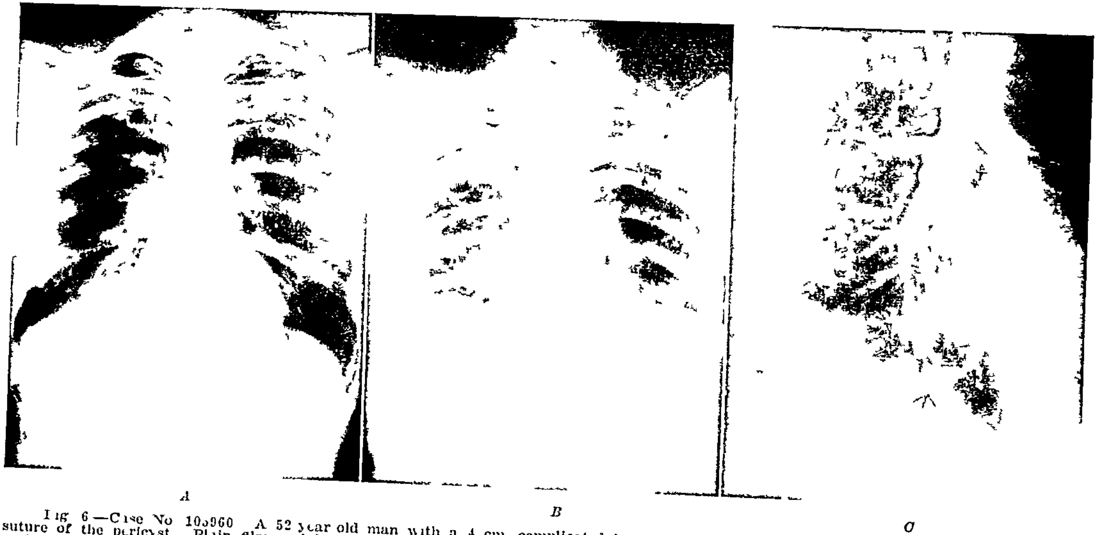


Fig 6—Case No 105960 A 52 year old man with a 4 cm complicated hydatid cyst in the right lower lobe Treated by internal suture of the pericyst Plain film and bronchogram 6 months postoperative A 1 5 cm residual cavity communicates with the right posterior basal bronchus Symptoms now consist of occasional nonproductive cough A, Preoperative B Postoperative C Bronchogram

are inevitably lost. The functional result in 26 followed patients is summarized in Table III. No patient had significant residual symptoms requiring treatment. We know of no recurrences due to secondary cyst formation.

TABLE III. FOLLOW-UP RESULTS ON 42 PATIENTS TREATED BY INTERNAL SUTURE

DURATION OF FOLLOW-UP	SIMPLE CYST		COMPLICATED CYST	
	EXCELLENT†	GOOD*	EXCELLENT	GOOD
<6 months	4		3	1
½-1 yr.	1			2
1-2 yr.	2	2	2	2
>2 yr.	3	1	2	1
Totals	10	3	7	6
No follow-up	9		7	

*Excellent—No symptoms.
†Good—Occasional minimal cough or chest pain.

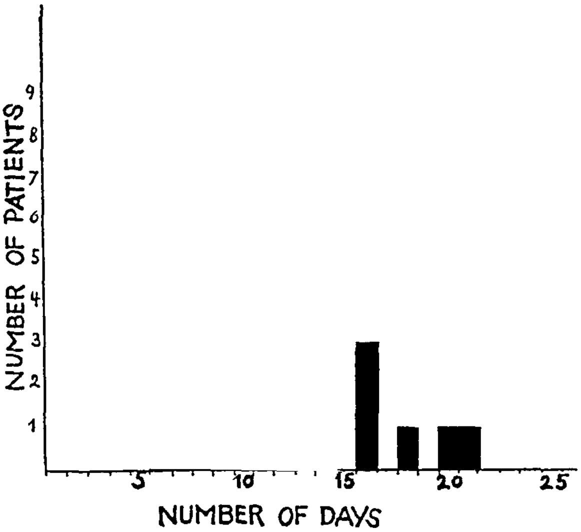


Fig. 5.—Duration of postoperative hospitalization after 46 thoracotomies for internal suture of hydatid cyst.

Anatomic results were checked in 20 of the 26 patients by bronchogram at a period varying from a few months to several years postoperatively. In 12 patients the cysts were simple and in 8 cases they were complicated. The findings follow: In a total of 20 patients there was no residual cavity or bronchiectasis in 17 patients; minimal residual localized bronchiectasis in 1 patient; and residual cavity, 1.5 cm. diameter (Figs. 6 and 7), in 2 patients.

In the 3 patients showing slight residual changes on bronchogram, the cysts were complicated and in the lower lobe. One of the patients (with residual 1.5 cm. cavity) has occasional cough and the other 2 have no symptoms.

In conclusion, our studies confirm that internal suture of the pericyst is safe and effective. We could wish for longer and more complete follow-up of our patients and hope by continuing observation to arrive at a more thorough evaluation of this problem in the future. The method is applicable to both simple and complicated cysts. Even large hydatids can be well obliterated as shown by Fig. 8. Fortunately, the procedure is technically easy and allows maximum preservation of functioning lung tissue.

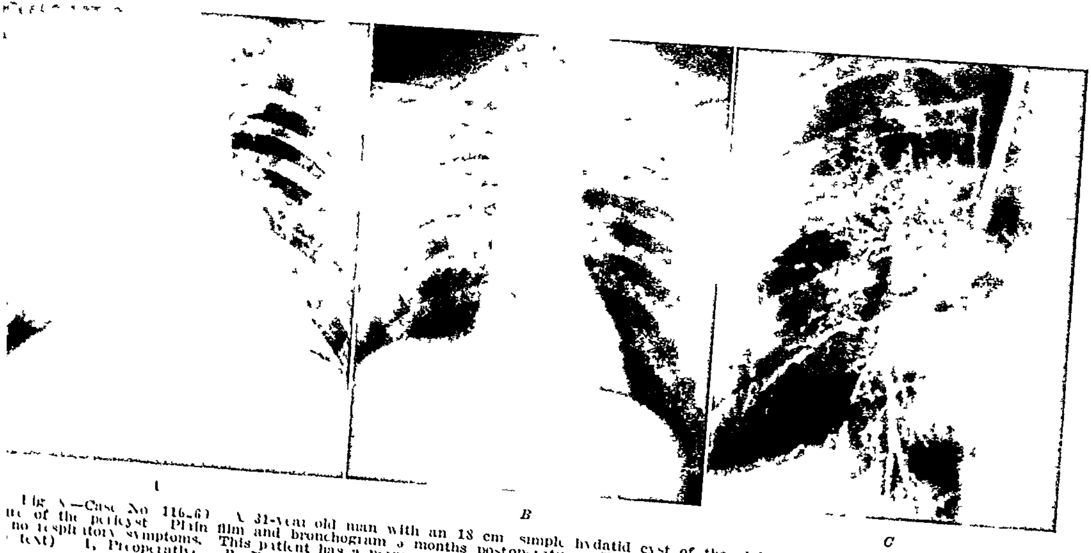


Fig. [illegible].—Case No. 116-61. A 31-year-old man with an 18 cm. simple hydatid cyst of the right middle lobe. Treated by internal ue of the pericyst. Plain film and bronchogram 3 months postoperative. There is no residual cavity or bronchiectasis. He now no respiratory symptoms. This patient has a permanent left hemiplegia presumably due to cerebral air embolism during operation text). A, Preoperative. B, Postoperative. C, Bronchogram.

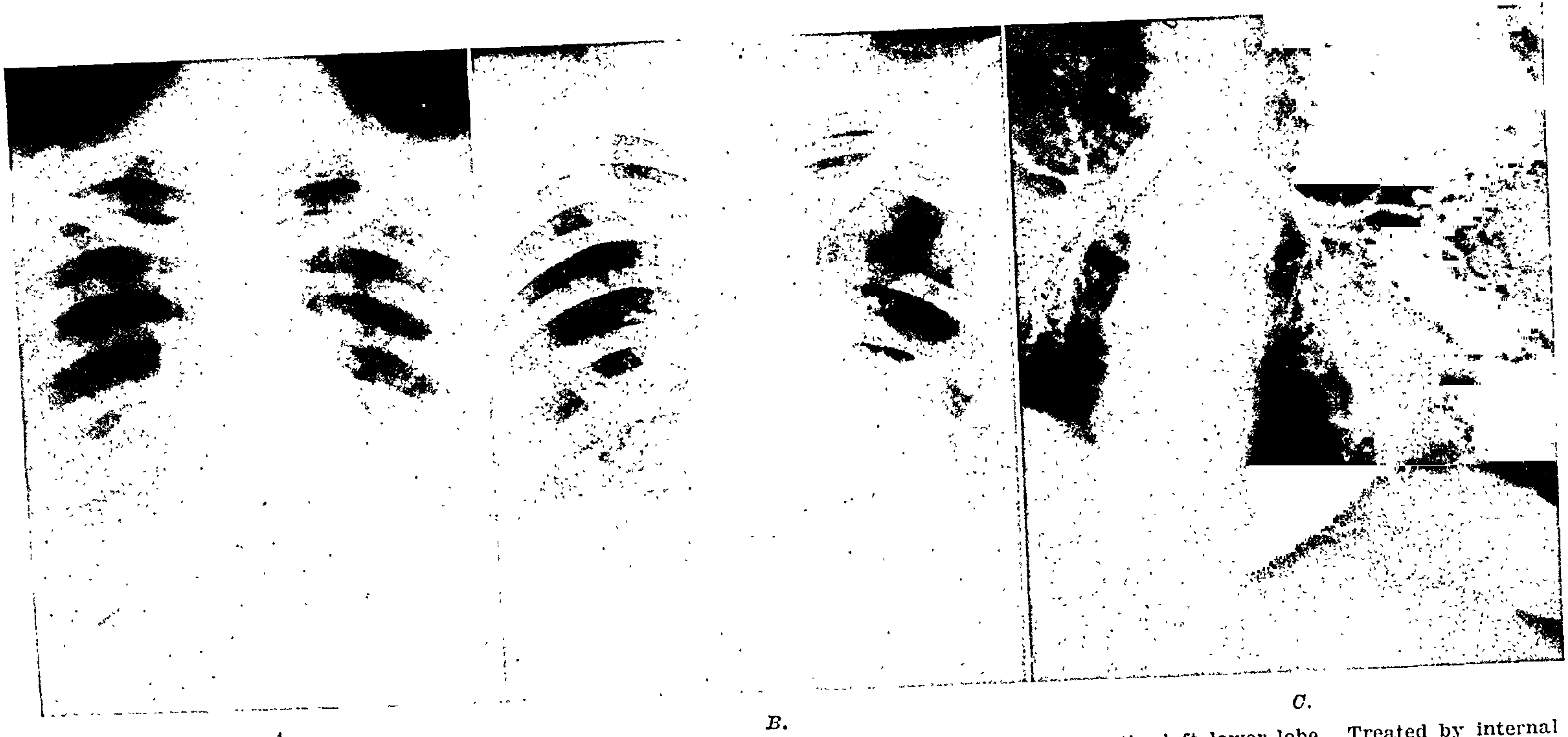


Fig. 7.—Case No. 62066. A 33-year-old woman with a 6 cm. complicated hydatid cyst in the left lower lobe. Treated by internal suture of the pericyst. Plain film and bronchogram 4 months postoperative. A residual 1.5 cm. cavity communicates with the posterior basal bronchus. She now has no respiratory symptoms. *A*, Preoperative. *B*, Postoperative. *C*, Bronchogram.

PANCREATIC PSEUDOCYST OF THE MEDIASTINUM

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INTRODUCTION

It is the purpose of this presentation to record the rare instance of pseudocyst of the pancreas presenting as a tumor of the mediastinum, and to discuss its diagnostic and therapeutic considerations

CASE REPORT

History—A 41 year old white, married, used tire dealer entered the hospital complaining of mid back pain and easy fatigability of 6 weeks' duration The pain was mild, recurred almost daily, lasted several hours at a time and was unrelated to meals or type of food ingested On some occasions exercise relieved the pain

At the onset of his illness 3 months earlier the discomfort was different He experienced sharp, anterior left chest pain radiating through to his mid back and to his shoulders It was more intense with deep breathing This pain disappeared in 24 hours An electrocardiogram taken at the time was normal There was no recurrence of this type of chest pain Later that month an attack of left flank pain was believed to have been the result of a renal calculus A stone was not found at cystoscopy

There were no complaints of cough, night sweats, fever, sputum, or hemoptysis There was no precordial pain with exertion and no symptoms involving the upper extremities He reported that he had lost 35 pounds over a period of 7 months

In reviewing the past history, the patient reported that 3 years earlier, after a night of heavy drinking, he had been admitted to another hospital with severe epigastric pain, nausea, and vomiting After x ray studies, emergency surgery was performed The discharge diagnosis was perforated peptic ulcer Following this procedure, he had had no gastrointestinal symptoms or recurrence of epigastric pain

His habits included moderate daily alcohol intake, primarily whiskey, 1½ packs of cigarettes daily, and no medications He reported no food intolerance or allergies The family history was noncontributory

Physical Examination—The patient was a well developed, well nourished, ambulatory, white male in no acute distress Blood pressure was 160/100 mm Hg, temperature, 99° F , pulse, 88, respirations, 20 Examination of the chest revealed a palpable friction rub over the left lower chest, anteriorly, along with tenderness to percussion over the tenth to twelfth ribs in the left midaxillary line This rub was not elicited after admission There were no other adventitious sounds The heart was normal The liver was not palpable There was a right upper quadrant scar The remainder of the physical examination was within normal limits

Laboratory and X ray Data—Detailed hematology, blood chemistry, urine and stool studies were normal with the exception of the sedimentation rate of 34 mm per hour

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SUMMARY

During the past 10 years, 62 patients with primary hydatid cyst of the lung have been treated surgically at the American University Hospital in Beirut without mortality.

Although a variety of procedures have been carried out, interest in recent years has been on treatment by internal suture of the pericyst after careful removal of the parasite.

Forty-two patients have had cysts treated in this manner with low morbidity and minimal complications. Follow-up examination of 26 of these patients disclosed that none had significant residual symptoms. Bronchograms on 20 of them showed that only 3 had either slight localized bronchiectasis or minimal residual cavitation.

Long-term follow-up of greater numbers of patients treated by internal suture is necessary before a thoroughly objective study of this method of treatment will be completed. However, on the basis of present evidence, it is justifiable to recommend internal suture as suitable for most hydatid cysts of the lung.

The assistance of Miss Batishwa Badawi, School of Public Health, American University of Beirut, is gratefully acknowledged.

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X-rays of the chest revealed a large, globular soft tissue mass in the lower mediastinum posteriorly, located above the diaphragm on both sides of the spine. Repeated chest x-rays proved that the mass was increasing in size (Fig. 1). Esophagrams were negative for intrinsic lesions and diaphragmatic herniae. There was deviation of the esophagus to the left without impingement upon its lumen. The gastrointestinal series showed the duodenum to be irritable with a pressure defect noted within the duodenal sweep which was in itself not enlarged. Angiography did not opacify the aorta sufficiently to delineate the vascular structures from the mass. However, thoracic aortography clearly showed the aorta to be deviated by the lesion. A bone survey was negative.

Operations.—The preoperative impression was cystic tumor of the posterior mediastinum. Exploratory thoracotomy was carried out through the bed of the right eighth rib. A supradiaphragmatic cystic lesion, 8 by 5 by 4 cm. was found to the right of the lower third of the esophagus. It was densely adherent to the posterior aspect of the pericardium, and a portion of the cyst extended below the diaphragm, through the aortic hiatus. The cyst contained thin, muddy-brown fluid, small gray amorphous particles, and an amylase content of 3,050 units. The supradiaphragmatic component of the pseudocyst was excised. The subdiaphragmatic extension was closed around a No. 18 Foley catheter by means of a purse-string suture. Urokon (30 per cent) was instilled through the catheter and was visualized in the region of the tail and body of the pancreas (Fig. 2). A diagnosis of pancreatic pseudocyst with mediastinal extension was made. Transduodenal sphincterotomy was decided upon. The thorax was closed in layers. The Foley catheter and intercostal tube were brought out in the midaxillary line.

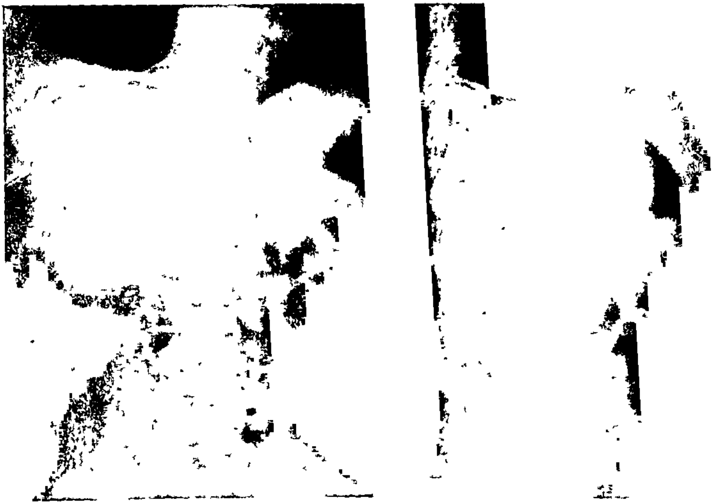

Fig. 3.—Retrograde pancreatogram taken on the fourteenth postoperative day ([A] antero-posterior and [B] lateral views).

The condition of the patient after turning him onto his back permitted abdominal exploration and definitive therapy. An ample upper abdominal right rectus incision was made. There was no free fluid in the peritoneal cavity. The gall bl [illegible] and

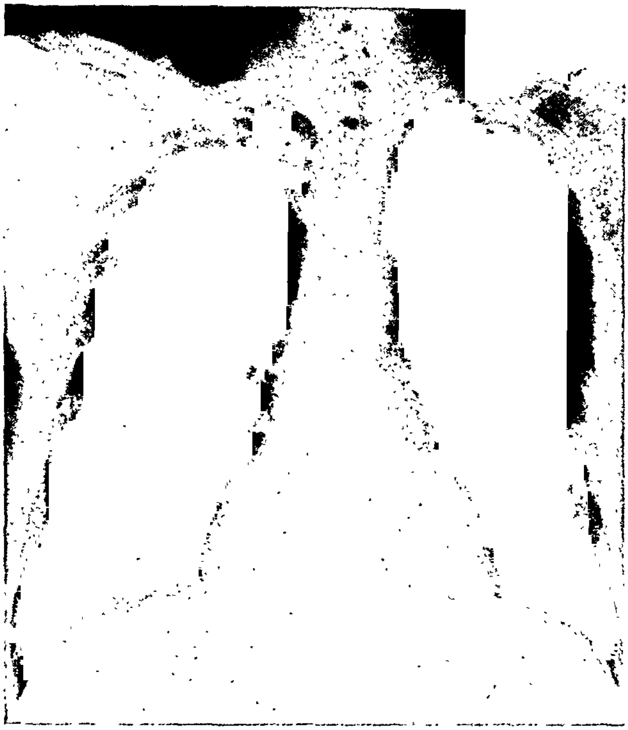

Fig. 1.—A preoperative chest x-ray demonstrating a mass in the posterior, inferior mediastinum.

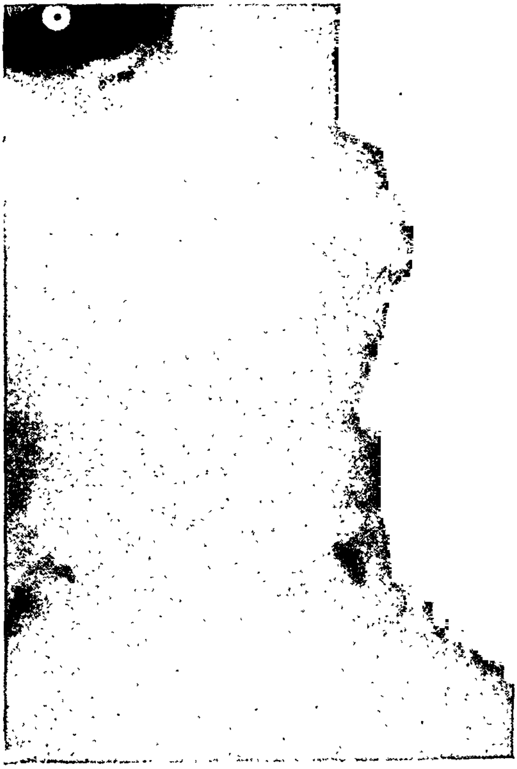

Fig. 2.—Retrograde pancreatogram, taken during right thoracotomy, illustrating the subdiaphragmatic portion of the pancreatic pseudocyst and the dilated duct of the tail and the body of the pancreas.

symptoms, if any, and the x-ray appearance of a dense rounded paraspinal mass with a sharp border and, rarely, bony erosion of the rib or enlargement of the intervertebral foramen. Esophageal neoplasms, epiphrenic diverticula,[2] and hiatus herniae frequently are suspected by symptoms and confirmed by barium meal contrast x-ray studies. They appear on the left side. Pneumoperitoneum may help differentiate diaphragmatic defects and herniation of abdominal viscera. Gastroenterogenous cysts,[3] duplications of the alimentary tract,[4] meningocele,[5] sequestered lung,[6] lipoma, pheochromocytoma,[7] and displaced kidney[8] are rare and tend to elude definitive diagnosis preoperatively, despite studies of known parameters.

Teratomas occur anteriorly[1] Pericardio-coelomic cysts[9] are rare, especially posteriorly. Tuberculous adenitis is usually in the hilar region. Tumors of lymphoid tissue are likely to have coexisting enlargement in the more classical locations The rare mesenchymal tumors (hemangioma, lymphangioma, pericytoma) defy diagnosis prior to histologic study. Aortic aneurysm usually appears on the left side and may be visualized with contrast media.[10, 11] Exotic hepatic infections[12] usually have abdominal findings. Lung tumors suggest the diagnosis by history and endoscopy.

Pseudocysts[1] of the pancreas are considered following the history of recurrent pancreatitis with epigastric and/or back symptoms, and an epigastric mass of inconstant or enlarging size. Pseudocyst of the posterior mediastinum is likely to elude diagnosis unless an abdominal component of the pseudocyst is recognized[1] Definitive diagnosis follows demonstration of pancreatic enzymes in the fluid and communication between pancreas and the mediastinal mass[1] The patient reported here lacked the classical history of pancreatitis and had no abdominal mass. The succession of chest pain, flank pain, and back pain may have indicated extensions of the pseudocyst.

Needle puncture of a mediastinal mass is ill advised if the *mass contains* tumor [illegible] d tissue, especially *when the possibility of transgressing the* pleura [illegible] *Injecting contrast media is not without hazard of extravasation* [illegible] *(e.g., into the subdural space). Aspirated fluid of high* amylase [illegible] *specific for pancreatitis. It has been found in pleural* effusions [illegible] *primary carcinoma of the lung, and retroperitoneal* primary and secondary *tumors invading the thoracic duct.*

When the nature of the mediastinal mass remains in doubt after appropriate studies thoracotomy is indicated for diagnosis and therapy.

THERAPY

If the diagnosis of pancreatic pseudocyst is made through preliminary studies, surgical treatment appears mandatory to reverse the process of enlargement of the pseudocyst, and to relieve symptoms.[1] In the present case, abdominal surgery would have been performed.

One philosophy of therapy advocates sphincterotomy[1] to eliminate the mechanical factors believed to be provocative of recurrent [illegible] *reatitis, aff*[illegible] *ing natural route internal drainage of the cyst by the* [illegible] duodenum were normal to palpation. The pancreas was harder and more nodular than normal. Exploration of the lesser sac revealed no evidence of a cyst. The Foley catheter could be palpated retroperitoneally. A transduodenal sphincterotomy was performed, followed by cholecystectomy. A polyethylene tube was threaded up the pancreatic duct and dye was injected for a pancreatogram. The pancreatogram was imperfectly performed. It was not possible to delineate communication between the cyst and the pancreatic duct system. The distal end of the polyethylene tube was then passed up the common duct and brought out through the cystic duct stump. A short limb T-tube was inserted into the common duct and brought out through a stab wound with the pancreatic duct catheter and with the Penrose drain placed at the foramen of Winslow before closing the abdomen in layers. During the 5-hour procedures, 1,200 c.c. of blood and 1,000 c.c. of 5 per cent glucose in water were given.

Fig. 4.—Chest x-ray one year after removal of mediastinal mass.

Postoperative and Follow-Up Courses.—The patient did well postoperatively. The pleural drainage tube was removed on the third postoperative day.

There was little drainage from the Foley (pseudocyst) catheter or from the pancreatic duct catheter. X-rays of simultaneous injections of contrast media through these catheters showed no residual pseudocyst, and no dilatation of the pancreatic duct system, as had been noted the day of surgery (Fig. 3). These catheters were removed. T-tube cholangiography showed no reflux into the pancreatic duct. The T-tube was removed.

The patient has been seen periodically for 14 months since surgery. His only symptom is fatigue. His appetite is normal. His diet is not restricted. His weight is steady at his normal 165 pounds. The physical examination is normal except for well-healed scars. His history, physical examination, and gastrointestinal x-rays give no indication of recurring pseudocyst (Fig. 4).

DIFFERENTIAL DIAGNOSIS

In the list of tumors of the posterior inferior mediastinum[1] are neurilemmoma and the less common ganglioneuroma, which are characterized by few

5 Byron, F X, Alling, E E, and Samson, P C Intrathoracic Meningocele, J THORACIC SURG 18 294, 1949
6 Brewer, A J, Clagett, O T, and McDonald, J R Intralobar Bronchopulmonary Sequestration, Am J Roentgenol 71 751, 1954
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14 Doubilet, H, and Mulholland, J H Pancreatic Cysts, Principles of Treatment, Surg, Gynec, & Obst 96 683 1953
15 Doubilet, H, Poppell, M H, and Mulholland, J H Pancreatography, Radiology 64 325, 1955
16 Doubilet, H Section of the Sphincter of Oddi, Surg Clin North America 36 865, 1956

forms of surgical therapy still enjoying popularity employ internal drainage by "cyst-enterostomy,"[13] or external drainage. A disadvantage of the latter is the electrolyte loss. The disadvantage of both may be the failure to obviate recurrent pancreatitis.

Technical considerations influence or compel certain surgical actions. *Excision may or may not be possible. The retroperitoneal portion of the pseudocyst* may be of insufficient size to permit enteroanastomosis. External drainage may be a mandatory safety valve.

DISCUSSION

In this reported case, excision of the bulk of the mediastinal mass was begun as definitive treatment but was abandoned when partial excision revealed the extent and identity of the pathology. External drainage was employed as a safety valve and as a pathway for postoperative dye injection studies. Sphincterotomy was elected to relieve the pancreatic duct obstruction believed responsible for recurrent pancreatitis and the pseudocyst. The absence of a palpable retroperitoneal mass precluded enteroanastomosis, had it been considered desirable. Cholecystectomy was performed prophylactically.[16]

All procedures were performed the same day, under the same anesthesia, to effect definitive treatment. The diagnosis appeared clear. It was believed that additional helpful data could not be obtained by other studies. The satisfactory condition of the patient before and during surgery, including turning the patient from the lateral to the supine positions, permitted this.

The immediate postoperative course, the 14-month sequel of well-being, and the radiographic evidence suggests that sphincterotomy was well chosen as therapy for this patient's problem.

SUMMARY

The case of a patient with a pancreatic pseudocyst of the lower posterior mediastinum is presented for consideration in differential diagnosis of symptomatic lesions of that area. The history and physical examination did not suggest the diagnosis.

Treatment consisted of transthoracic excision of the bulk of the cyst, and transduodenal sphincterotomy through a separate abdominal incision. Abdominal operation for internal drainage would have been performed if the nature of the lesion had been known preoperatively.

The differential diagnostic possibilities and methods have been discussed. The nonspecificity of fluid amylase content is emphasized.

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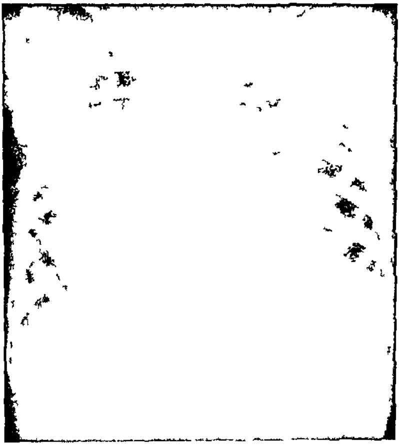
Fig 1

Fig 2

Fig 1 —Preoperative posteroanterior roentgenogram of the chest
Fig 2 —Preoperative lateral roentgenogram of chest

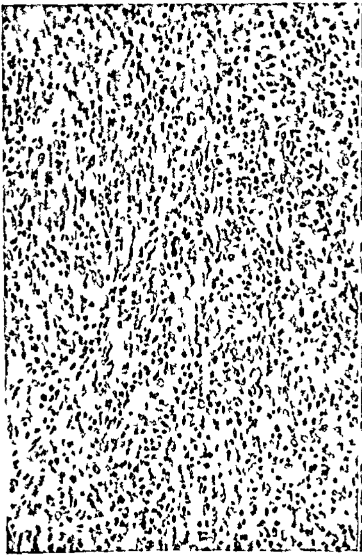
Fig 3

Fig 4

Fig 3 —Low power view showing proliferative fibroblasts with an admixture matory cells and blood vessels
Fig 4 —A more detailed view of the pleomorphic lesion

POSTINFLAMMATORY "TUMOR" OF THE LUNG

REPORT OF A CASE SIMULATING MALIGNANT THYMOMA

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ACUTE lower respiratory infections are occasionally harbingers of serious intrathoracic disease. Thus, many otherwise silent and unsuspected pulmonary lesions become available to roentgenologic scrutiny. There is, however, a rather rare group of postinflammatory lesions of the lung, aptly described by Umiker and Iverson,[1] which is easily confused both clinically and pathologically with neoplasm.

Herein is reported a case which presented as a rapidly growing neoplasm of the upper lobe of the right lung and was considered a malignant tumor on the initial pathologic examination.

CASE REPORT

A 3½-year-old boy was considered well until March 25, 1956, at which time he became acutely ill with a productive cough accompanied by a temperature elevation between 102° and 103° F. Treated initially as an outpatient, he was admitted to a Station Army Hospital on March 29, 1956, because of continued symptoms. Admission roentgenogram of the chest revealed a large dense shadow localized to the upper one-third of the right lung. He was diagnosed as having pneumonia and treated with penicillin and streptomycin.

Physical examination revealed an acutely ill child with a productive cough, fever of 102° F., and rather enlarged submaxillary and axillary lymph nodes. There were dullness and tubular breath sounds over the upper anterior and lateral portion of the right chest. Initial antibiotic therapy was considered ineffective. Bone marrow and routine laboratory studies were not helpful diagnostically. He was therefore transferred to Fitzsimons Army Hospital on April 3, 1956, for further evaluation and treatment.

The physical findings on transfer revealed a pale, acutely ill boy, coughing frequently and productively, with a temperature of 99.8° F. There were diminished breath sounds over the right anterior chest with slight but significant bulging in this region. No mediastinal shift was observed.

Chest roentgenograms taken at Fitzsimons Army Hospital and compared with those previously taken revealed an enlarging density in the anterior right chest continuous with the anterior superior mediastinum (Figs. 1 and 2). Diagnostic intravenous urogram showed no abnormalities. The admission blood count showed 6,800 white cells with 70 per cent neutrophils; hemoglobin 10 grams; hematocrit 31; and the sedimentation rate was 53.

It was the impression of the staff that this patient had a rapidly growing malignant neoplasm involving a large part of the right upper pulmonary lobe and upper anterior mediastinum. Therefore, after blood transfusion, a right exploratory thoracotomy was performed on April 7, 1956, at which time a large firm lobular tumor was found extending from the superior and anterior mediastinum into the right upper lobe which it largely replaced.

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This material has been reviewed and there is no objection to publication by the Office of the Surgeon General, Department of the Army, MEDCI, June 19, 1957.

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days to May 17, 1956 The patient received 6 treatments a week, using 250 kv, directed through 5 by 6 cm anterior and posterior fields covering the hilar areas and the superior mediastinum Alternate fields received 200 r air daily for a total air dose of 2,800 r per field The skin dose was 3,780 r, and estimated tumor dose was 2,684 r Fourteen treatments were given to each field The skin reaction was mild on the completion of therapy

The patient was followed at regular intervals The last visit was one year after surgery at which time he was entirely free of evidence of recurrence, was in excellent health, and growing normally (Fig 5)

Final Pathologic Diagnosis—Following reconsideration of the pathologic specimen, the diagnosis originally entertained was replaced by that of "postinflammatory tumor of lung and anterior mediastinum "

DISCUSSION

Postinflammatory tumors of the lung as previously described, are characteristically solitary, circumscribed, and of uniform density varying from 2 5 to 7 cm in diameter [1, 2] They have been located in the parenchyma of the lung in the majority of cases but have been found in the pleura,[3] mediastinum,[2, 4] or been present in all 3 locations as in our case Previous recurrent or concurrent lower respiratory infections have been present in most patients Acute febrile pulmonary infections progressing concomitantly with such tumor formations are relatively rare Umiker and Iverson describe but 2 of their 7 cases as occurring in this manner Other cases have had free intervals of from 2 months to 22 years before the pulmonary lesions were found and identified

The age distribution has varied from 3 to 61 years [2, 5]

Evidence for the benignity of these pseudotumors has been their failure to recur The youngest patient has lived 8 years without reappearance of the tumor

In seems probable that an unresolving pulmonary or mediastinal density or one enlarging, as in our case, immediately following an acute lower respiratory infection, especially in the younger age group,[6] is an important clinical feature and should lead one to suspect a postinflammatory tumor

The histologic features of these lesions include a proliferation of fibroblasts with or without production of conspicuous collagen, formation of numerous small vascular channels, and an admixture of inflammatory cells, chiefly plasma cells Variable degrees of hemorrhage may occur and foam cells may become prominent In reported cases there is a conspicuous lack of mitotic figures

Because of the pleomorphic pattern and the quantitative variability of the components, various diagnoses have been applied to these lesions They have frequently been mistaken for sarcomas [1, 7] Diagnoses of fibroma, fibrous xanthoma or xanthoma have been entertained when the appropriate components predominate [8, 9] It would appear that the quantity of xanthomatous elements are more closely related to the degree of hemorrhage than other factors, thus producing a tissue response similar to that observed in a dermatofibroma of the cutis When plasma cells are a feature, the differentiation from a plasma cell neoplasm may be difficult Many of the plasma cell granulomas of the lung and mediastinum as reported in the literat [illegible] uld [illegible] ear to b

There were numerous succulent nodes about the hilus and frozen section of these revealed no tumor. Frozen section of the tumor, however, was interpreted as showing a malignant neoplasm. Grossly, it was possible to remove the entire tumor together with the right upper lobe and most of the thymus which appeared to be involved in the pathologic process.

Pathologic Report.—Grossly the tumor mass measured 10 by 7 by 5 cm. The specimen was rubbery and, upon cut section, exhibited a uniform grayish-white granular appearance with small inconspicuous areas of superimposed hemorrhage. Extension had occurred through the pleura from the parenchyma of the right upper lobe and irregularly into the mediastinal connective tissue. The thymus gland was immediately adjacent to the tumor but, grossly uninvolved. Slightly enlarged regional nodes presented a normal pattern.

Microscopically, the tumor was composed predominantly of a proliferation of closely situated spindle cells with oval, vesicular nuclei arranged in irregular interlacing strands or whorls. In more cellular areas, inconspicuous fine collagen fibers and numerous but inconsistent capillaries were formed. Infrequently, production of dense collagen was seen in areas of lesser cellularity. An admixture of plasma cells, lymphocytes, and neutrophils were scattered throughout. Many of the plasma cells were immature as evidenced by enlargement of their nuclei, a homogenicity of nuclear chromatin, and loss of the eccentric position of these nuclei. A reticulum stain exhibited moderate numbers of wavy fibers which were occasionally split longitudinally, showed no lateral branching, and passed between cells. Mitotic figures were rare, occurring approximately one per high-power field. Masson's trichrome stain presented an equivocal reaction. Russell bodies were not identified, and foam cells were absent (Figs. 3 and 4). The pathologic diagnosis of a thymoma was entertained.

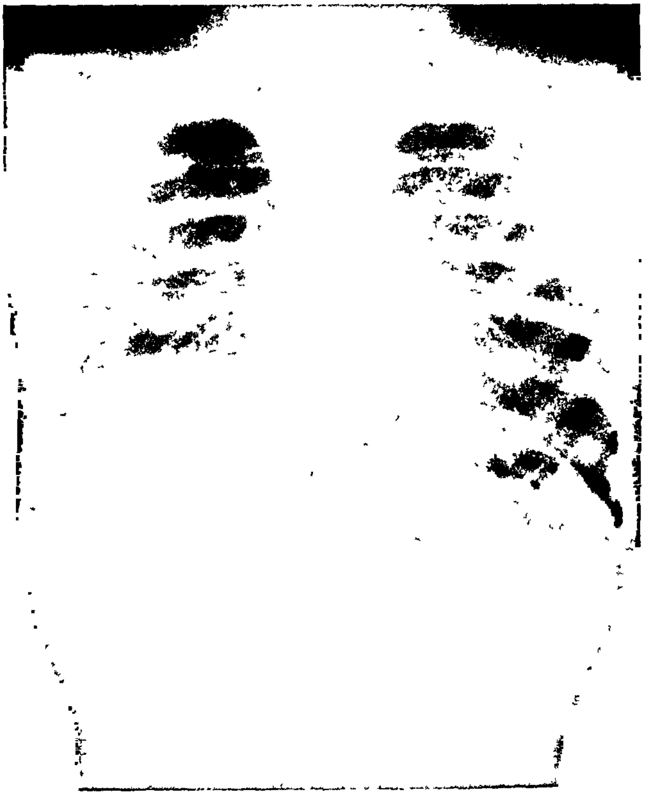

Fig. 5.—Postoperative posteroanterior roentgenogram of the chest one year after operation

Postoperative Management and Course.—The patient made an uneventful recovery. Because of the initial pathologic diagnosis and upon the recommendation of the Tumor Board, a course of x-ray therapy was begun on April 16, 1956, and extended over a period of 31

SOLITARY BRONCHIAL POLYPS OF INFLAMMATORY ORIGIN

A Report of Two Cases Treated by Operation

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Polyps are commonly found in the nose, the larynx, and the trachea. A solitary polyp in the bronchus is, however, very rare. Bednář,[2] Fourestier and associates,[4] and Šálek and co-workers[9] reported isolated cases and Lecoeur (cit. Fourestier) emphasized that in some thousands of bronchoscopies he had met with only one case. This low rate of incidence is confirmed in our clinic where, during the last 10 years, we have had only 2 cases of solitary bronchial polyp.

CASE REPORTS

Case 1.—A. J., a 61 year old worker, stated that he had always been healthy, but that 10 years previously an irritative cough had developed with a small quantity of mucous expectoration. X-ray examination carried out 5 years later did not show any significant abnormality in the lung. About 3 years before admission, he developed pain in the back and the x-ray examination at that time showed a chronic bronchitis, emphysema of the lungs, and pleurisy with a small effusion in the right costophrenic angle. He had recently been losing weight. No hemoptysis had occurred.

X-ray examination on admission in 1952 showed an opacity in the right supradiaphragmatic field due to atelectasis of the middle and lower lobes of the right lung.

Bronchoscopy revealed in the bronchus intermedius a smooth, globular, pink, soft, easily movable tumor, the size and shape of a small cherry. A biopsy was taken and showed on histologic examination, only fragments of bronchial mucous membrane and inflammatory exudate.

A right thoracotomy on July 18, 1952, disclosed atelectasis of the middle and lower lobes with numerous adhesions to the parietal pleura. In the hilus, soft, moderately enlarged lymph nodes were found. The middle and lower lobes were resected and, when the bronchus intermedius was incised, a pink, lobular, soft tumor, the size of a cherry, protruded from its lumen.

The immediate postoperative course was uneventful and on review, 4 years after operation, he appeared to be perfectly well.

The examination of the specimen revealed a mass, 2 by 1.5 by 1.2 cm., inside the bronchus intermedius where it was attached by a relatively large pedicle to the inner wall of the bronchus close to its origin and nearly obliterating its lumen. The surface of the tumor was papillary, some of the papillae were rugged, cauliflower like, while others showed smooth and globular form (Fig. 1). The tissue of the middle and lower lobes was the seat of chronic nonspecific pneumonia and was partly necrotic.

Histologically the tumor had a venous and fibrous stroma with some elastic fibers. In its central portion, deposits of fatty tissue were found (Fig. 2). The stroma was permeated by lymphocytes and plasma cells, especially near the surface of the papillae. The

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included in the subject under discussion.[2, 4, 5, 10] Some of these have involved lymph nodes as well as pulmonary tissue with partial or complete replacement of the nodal architecture. They may contain abnormal and juvenile forms of plasma cells indistinguishable per se from cells of multiple myeloma.[11] The relationship of the plasma cell granulomas of the mediastinum and lung to plasma cell lesions of the upper respiratory tract is still not completely settled. The true plasma cell neoplasms consist of a solid growth of plasma cells without inflammatory phenomena of vascularity, collagen production, and infiltration of varieties of other inflammatory cells.[12] The presence of Russell bodies in inflammatory lesions and their absence in neoplastic lesions has not proved a reliable criterion of differentiation.[11] None of the plasma cell granulomas presenting primarily in the lung or mediastinum have developed multiple osseous lesions of multiple myeloma.

SUMMARY

A clinical case of a 3½-year-old white boy is reported with the diagnosis of postinflammatory tumor of the lung and mediastinum. The gross appearance and extension of the process in the upper lobe of the right lung to the anterior mediastinum, in our opinion, gave ample justification for the extirpative management of the poorly delineated "tumor" which was initially diagnosed pathologically as being a malignant thymoma. Postoperative deep x-ray therapy, approximating that amount usually given in the treatment of radiosensitive malignant neoplasms of the lung and mediastinum, was given prior to arriving at the apparently correct pathologic diagnosis. (Irradiation therapy is not recommended for such "pseudotumors".) The patient is growing normally and is asymptomatic without roentgenographic evidence of residual pulmonary, pleural, or mediastinal involvement over one year after surgical removal of this uncommon and poorly understood chronic inflammatory lesion.

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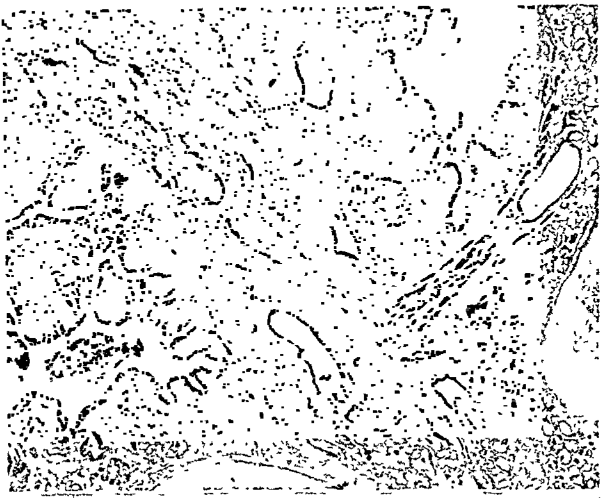
Fig. 2.



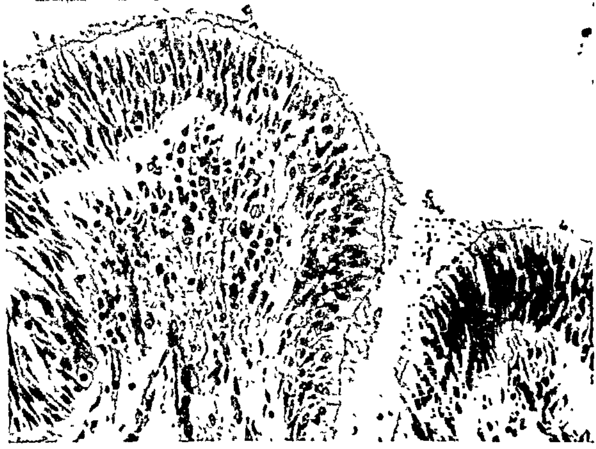


Fig. 3.

Fig 2.—Case 1 The pedicle of the bronchial polyp (hematoxylin-eosin-elastic; ×22).

Fig 3.—Case 1. The surface of polyp covered with stratified ciliated columnar epithelium (hematoxylin-eosin; ×290).

surface of the tumor was covered with ciliated columnar epithelium (Fig. 3). The wall of the bronchus to which the tumor was attached was diffusely affected by chronic inflammatory changes.

The bronchial glands were distinctly enlarged.

The histologic findings led to the conclusion that this was a case of extreme hyperplasia of bronchial mucous membrane of inflammatory origin.

Case 2.—In A. F., a 36-year-old blacksmith, there developed, 5 years before admission, a dry cough with some mucous expectoration, followed in 1952 by dyspnea and pain in the left chest. X-ray examination in 1954 revealed only a slightly increased opacity of the left lung. Bronchoscopy, carried out in a sanatorium in 1955 demonstrated a tumor in the left main bronchus. A fibroma or neurofibroma of the bronchus was suspected. With this diagnosis the patient was admitted to the hospital. In 8 or 9 months prior to admission he had lost 8 Kg. in weight. There had been no hemoptysis.

X-ray examination, tomography, and bronchography revealed a slight diffuse opacity of the whole of the left lung, caused by a partial obstruction of the main bronchus by a tumor within the bronchus.

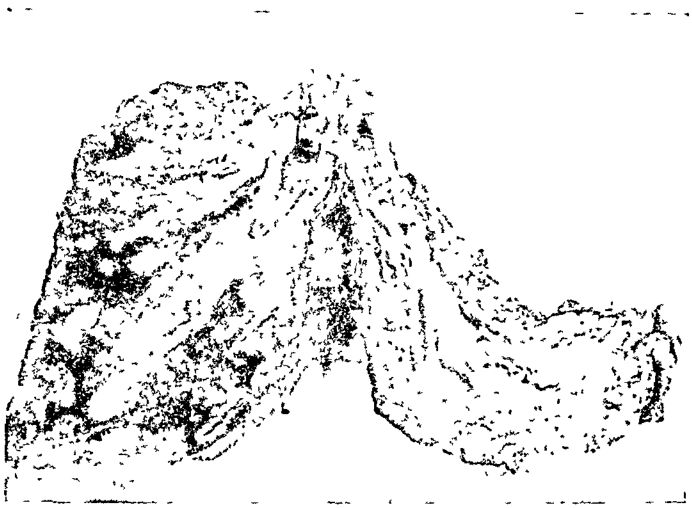


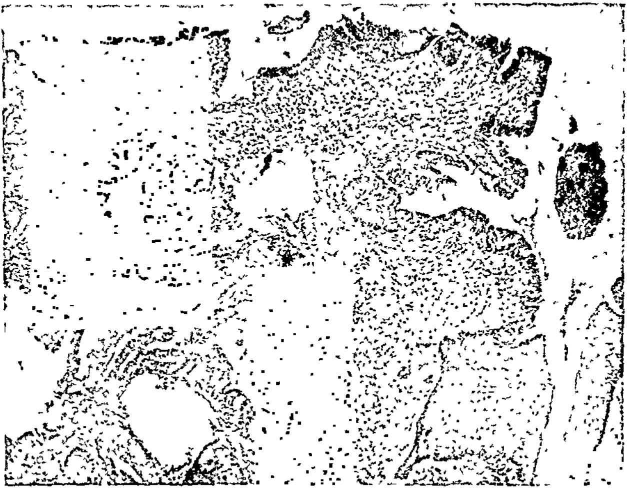
Fig. 1.—Case 1. The middle and lower lobe of the right lung. The lumen of resected bronchus is closed by the inflammatory bronchial polyp.

Bronchoscopy revealed in the left main bronchus, about 2 cm. below the carina, a smooth, globular, pedunculated tumor, the size of a cherry, attached to the posterior wall of the bronchus. Macroscopically, it appeared to be a lipoma.

Histologic examination of the bronchoscopic specimen, consisting of a part of the hypertrophic bronchial mucosa (Fig. 4), showed that to a large extent it was covered on the surface with proliferating, evidently metaplastic squamous epithelium. The lamina propria of the mucosa was very thick and edematous, containing some isolated, foamlike macrophages. In some places the interstitial tissue gave a slightly positive histochemical reaction for mucus. The histologic findings in the bronchoscopic specimen suggested an inflammatory polyp originating in bronchial mucosa with myxoid degeneration.

At operation on July 7, 1955, a slight diminution in aeration of the left lung was found. In the left main bronchus, a soft tumor was palpable. Bronchotomy was carried out and an oblong, pink, lobular, well-circumscribed tumor was found, which was attached to the wall of the bronchus about 1 cm. above the origin of the left upper lobe bronchus by a slender pedicle about 3 mm. in diameter. The tumor nearly closed the lumen of the main bronchus and extended into the upper lobe bronchus. The pedicle was excised together with its bed, the mucus was aspirated from the bronchial tree, and the bronchus sutured. The lung re-expanded very well.

Fig. 6.



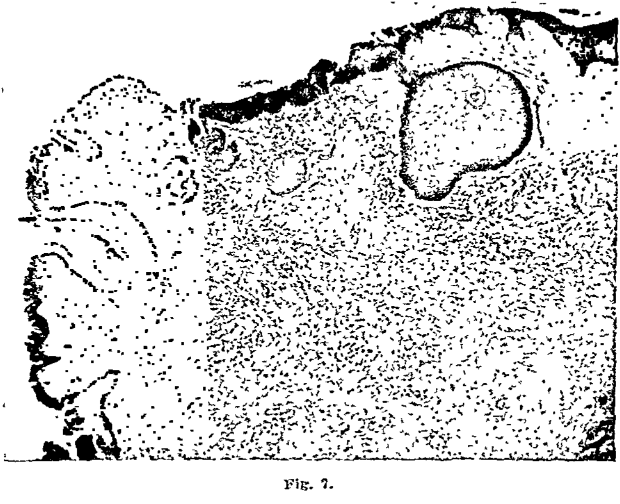


Fig. 7.

Fig. 6.—Case 2. The bronchial polypus is covered with columnar and squamous epi-[illegible]um (mucicarmine; ×50).

Fig. 7.—Case 2. The columnar and squamous epithelium on the surface of polypus [illegible]atoxylin-eosin; ×50).

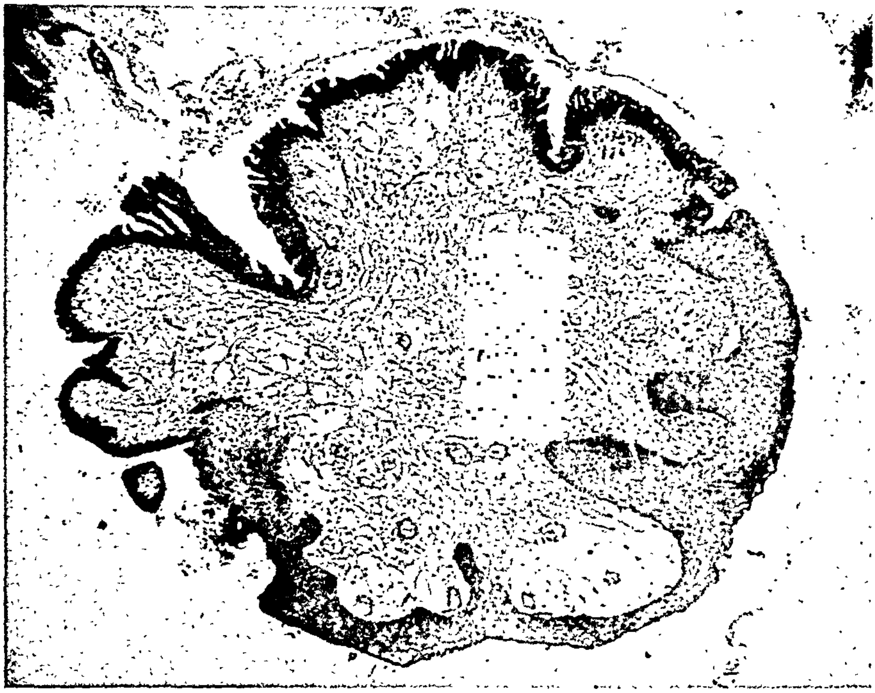


Fig. 4.—Case 2. The fragment of bronchial mucosa removed at bronchoscopy (hematoxylin-eosin; ×40).

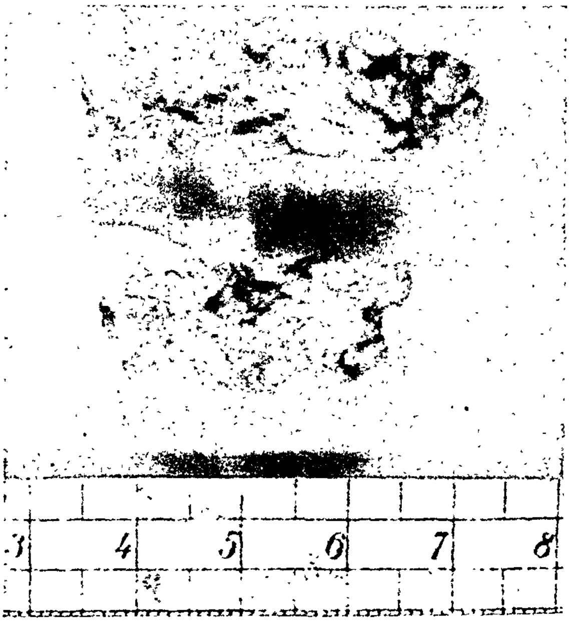


Fig. 5.—Case 2. The dissected polypus removed by bronchotomy.

The patient recovered and a subsequent bronchoscopy demonstrated adequate patency of the main bronchus, there was no recurrence of the tumor

The tumor measured 40 by 11 by 12 mm (Fig 5) and was of a grayish white color, with a rough surface, and of soft consistency with pink red deposits on the cut surface

Microscopic examination revealed a sparse, fibrous stroma which on the surface was covered partly by ciliated columnar epithelium (Fig 6), but mostly by metaplastic, stratified, squamous epithelium (Fig 7) Some parts of the stroma contained large numbers of fine collagenous fibers with small groups of siderophages Around the dilated veins there was a round celled inflammatory infiltration (Fig 8) Some sections were very edematous and, in places, signs of myxoid changes were evident The covering epithelium contained numerous goblet mucus cells The epithelium was marked by a striking proliferation in the form of solid columns of stratified squamous cells or in the form of closely connected papillary or cystopapillary formations (Fig 9) Although the proliferation of the epithelium was considerable, no abnormal cell types or atypical mitoses were found The process had no malignant character

In the lymph glands there was anthracosis and chronic nonspecific inflammatory reaction

Histologically, it appeared to be a case of a circumscribed polypoid hypertrophy of bronchial mucosa with squamous metaplasia caused most probably by a chronic inflammation

DISCUSSION

Bronchial polyps because of their rarity seldom come to the attention of the clinician and then not until their secondary effects are evident More frequently, they are seen by the pathologist at necropsy as a variant of the slight polypoidal hypertrophy of the mucosa which macroscopically need not look like a polyp The histologic structure of both forms is, however, analogous (Bednar[2]) The polyps are usually located in the main bronchus or its principal branches, either close to their origin or directly at the bifurcation of the bronchus Lecoeur maintains that this location is characteristic for inflammatory polyps

Clinically large polyps manifest themselves in the same manner as benign tumors of the bronchus Their symptomatology depends in many respects not only on the size of the polyp and on the diameter of the bronchus, but also on the character of the underlying inflammatory process Jackson and Jackson[7] contend that two symptoms are always present, hemoptysis and wheezy breathing, the latter particularly when the large bronchi are affected Neither of our 2 patients presented these symptoms, but each had had a cough for 10 and 5 years, respectively, no doubt a manifestation of chronic inflammatory changes in the bronchial mucosa which were seen in Case 1 From the histology we could not, however, ascertain the cause of this bronchitis Clinical symptoms attributable to the polyp manifested themselves in these 2 cases much later than the symptoms of bronchitis, 7 and 2 years, respectively, from the onset of persistent cough These clinical features, however, are in no way pathognomonic, for correct diagnosis can be made only by bronchoscopic biopsy

Macroscopically, these polyps are circumscribed masses of a globular or oval shape, attached by a pedicle to the inner wall of the bronchus Their rface is smooth but lobular, sometimes velvety or even rough and, s

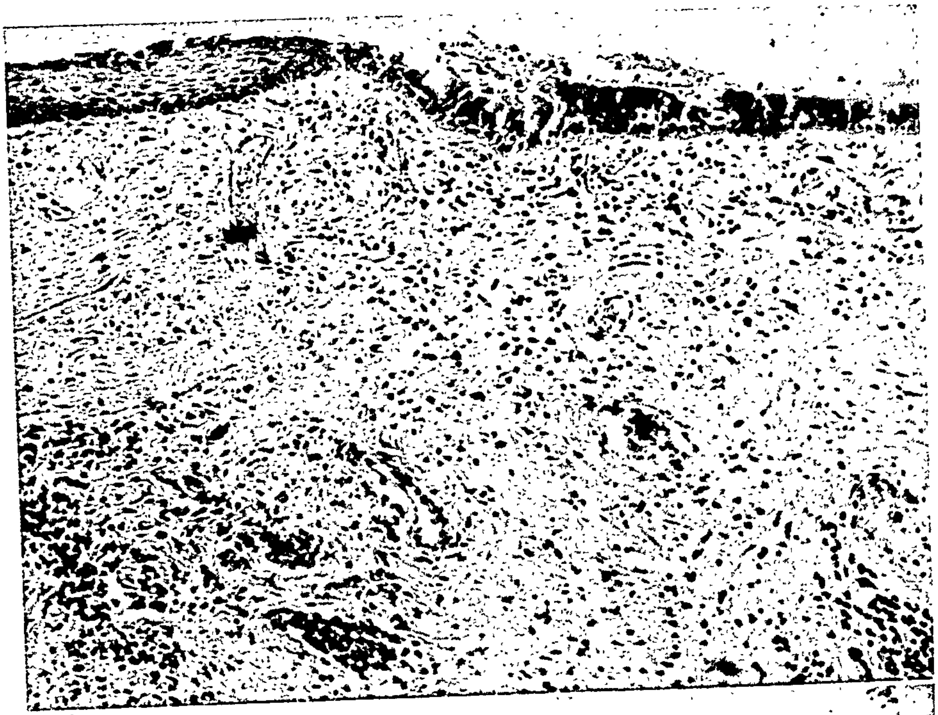


Fig. 8.

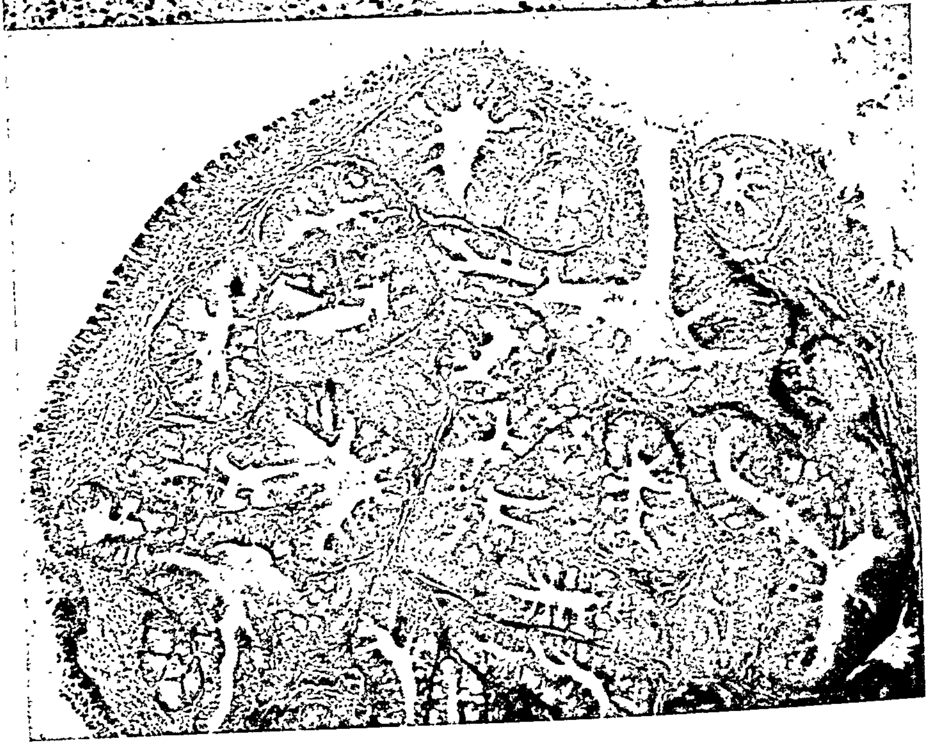


Fig. 9.

Fig. 8.—Case 2. A round-celled inflammatory infiltration of the stroma (hematoxylin-eosin; ×115).

Fig. 9.—Case 2. Some details from the polypus simulating the glandular structures (van Gieson; ×40).

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ulcerated (Jackson[7]). Their color as seen by us at bronchoscopy and at operation is light pink or reddish and the polyp is frequently veiled by a fine covering of mucus. They are relatively friable.

The question remains whether polyps should be classed as real tumors or as examples of inflammatory hyperplasia. Histologic structure of the polypus, particularly when it is marked by papillomatous arrangement which we have seen in both the above cases, makes it necessary to consider a neoplastic process both histogenetically and in the differential diagnosis. In support of this possibility is the fact that, in some cases, the inflammatory cellular infiltration of the stroma is slight and sometimes even the squamous metaplasia of the covering epithelium is not present (Case 1). Therefore, some authors (Jackson,[7] Hollmann,[6] Lindgren[8]) classify these polyps as benign bronchial tumors, perhaps analogous to fibro-epithelial neoplasm in other locations, as for instance condyloma acuminatum (Bednář), cutaneous papilloma, uterine mucous polyps, etc. The nature of these conditions is, however, also doubtful.

The relation of bronchial polyps to diffuse segmentary hypertrophy of the bronchial mucosa, which was described by Horányi[5] under the term "bronchial adenosis," is not yet clear. In about 3 per cent of 300 resected lungs, Horányi found circular stenosis of the bronchial lumen caused by hypertrophy of the mucosa with a striking increase and cystic enlargement of bronchial glands. He characterizes the process as a non-neoplasmatic hyperplasia but contests its inflammatory origin. A similar observation was made by Eszipova[3] in one case. We assume that inflammatory polyps can be a component part, though rarely, of chronic hypertrophic bronchitis. The inflammatory nature of this process is also admitted by Fourestier[4] and Bariety and his associates.[1]

We are of the opinion that polypoid hypertrophy of the bronchial mucosa is caused by a similar process as is operative in the development of mucous polyps in the nose. An inflammatory process, even localized, can produce a secondary narrowing of the lymphatic vessels and thereby a condensation of tissue fluid in the affected part of the polypoid hypertrophic mucosa. That is also the causative process in circumscribed edema of the mucosa, according to the observations of Bariety. Such an explanation would be sustained especially by the histologic appearance of the above reported Case 2. The question of the infective agent which brings about this inflammatory process remains unsolved. It seems that any inflammatory condition in the bronchus accompanied by a stagnation of the secretion, particularly when purulent, can be the decisive factor.

SUMMARY

Two cases of solitary inflammatory bronchial polyps which clinically manifested themselves by a cough of long duration and by later obstruction of the bronchi are reported. Both patients were treated by operation, one by lobectomy and the other by bronchotomy.

The authors discuss the genesis of these polyps and express the opinion that they are caused by chronic inflammation of the bronchial mucosa.

Fig 1.—Case 1. Mass in lower left pulmonary field partially obscured by left cardiac border

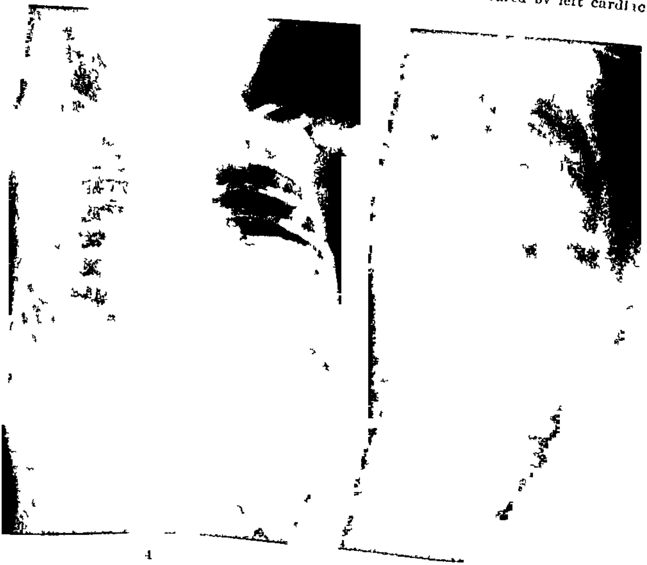

Fig 2.—Case 1. A, Film [illegible] months later showing [illegible] pronounced increase in size and [illegible]ity of mass. B, lateral view showing posterior location with [illegible] portion [illegible] shadow

HAMARTOMA OF THE LUNG

REPORT OF ONE MALIGNANT AND THREE BENIGN CASES

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ALTHOUGH the number of reported cases of pulmonary hamartoma is steadily increasing, there are few instances of reported enlargement of the tumor while the patient is under observation. The presence of malignant changes in these tumors is likewise rare. A case of a rapidly enlarging malignant hamartoma in the left lower pulmonary field of an infant, 22 months old, is the primary reason for this report. Three additional cases of benign pulmonary hamartoma are also included. These 4 cases were discovered at the center for Thoracic Surgery established by the United States Army Medical Service in Europe, between March, 1954, and March, 1956.

CASE REPORTS

CASE 1.—A baby, 22 months old, was first seen in August, 1954, because of a nodular shadow in the lower left pulmonary field discovered in a routine roentgenogram of the chest (Fig. 1). The patient had no thoracic symptoms and a decision was made to have her return later for further study. When the child returned in March, 1955, she still had no thoracic symptoms, and the only positive physical findings were increased breath sounds and dullness in the left lower pulmonary field, and definite Mongoloid features and retarded development.

Considerable increase in the size of the lesion was noted in roentgenograms of the chest; in fact the entire lower half of the left pulmonary field was obscured by the tumor (Fig. 2, *A*). The upper border of the mass was convex and smooth in outline. The heart and mediastinum were displaced to the right. In the lateral view the mass could be seen lying posteriorly with the anterior border reaching to the mid-portion of the cardiac shadow (Fig. 2, *B*). At fluoroscopy, the mass moved freely with respiration and could not be separated from the diaphragm. This led the observers to conclude that the lesion originated not from the mediastinum but rather from pulmonary tissue, and was possibly an enlarging bronchogenic cyst or teratoma.

Left lower lobectomy resulted in removal of the entire tumor. Recovery was uneventful and the patient was discharged in April, 1955. In March, 1957, there was no evidence of recurrence of the tumor.

The specimen was a lobe of lung containing a well-defined and circumscribed, nonencapsulated, fleshy, creamy white, trabeculated, solid tumor, 8 by 8 by 5 cm., with over two thirds of its surface covered by a thin pleura and the remainder of its margin attached to the adjacent compressed pulmonary parenchyma. The pleural surface was smooth and portions of the pleura could be stripped from the tumor. Where the tumor was adjacent

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Microscopic examination revealed a complex picture of multiple papillary processes and cystic spaces set in a stroma with a thin and indefinite capsule surrounding the whole mass. Many areas of fatty degeneration were present and in a few places the structure suggested cartilage of a primitive type. The lesion was considered to be a hamartoma but its superficial resemblance to a mixed tumor was noted.

CASE 3.—A white man, 35 years old, was found to have a nodule in the left pulmonary field on routine roentgenography of the chest on admission to the hospital. He had no thoracic symptoms. In the lateral view the tumefaction was located in the area corresponding to the lingula. Definite calcification could not be demonstrated in planigrams. At thoracotomy, the nodule was easily shelled out and the patient made an uneventful recovery. Cross section of the gross specimen showed the nodular, glistening appearance of the mass when freshly cut (Fig. 3).

CASE 4.—A white man, 47 years old, had evidence of a nodule, 1 cm. in diameter, in the right upper pulmonary field on routine roentgenography of the chest. Since no calcium could be identified within the lesion, removal was advised. The excised tumor, measuring 1.0 by 1.2 cm., was rounded, slightly lobulated, white, and firm. Sections showed a disorderly arrangement of cartilage, fat, fibrous tissue, and myxoid stroma. In this matrix were numerous, poorly formed bronchi, many of which showed a papillary configuration.

DISCUSSION

Albrecht[1] is generally credited with originating the term hamartoma in 1904. Since then, tumors containing an abnormal mixing or arrangement of the normal components of an organ have usually been designated as hamartomas. However, in the past, many tumors that were found in the pulmonary fields and were called chondromas, enchondromas, lipochondromas, lipo-osteo-adenochondromas, or whatever tissue combination existed in disorderly arrangement, were undoubtedly hamartomas. McDonald and associates[2] pointed out that many tumors reported as chondromas in the older literature were actually hamartomas.

At times the arrangement of the tissue components is such that classification is difficult. Two of the cases presented by Lemon and Good[3] were atypical in histologic appearance. Freedlander and associates[4] reported a case in which there was disagreement among the pathologists concerning the diagnosis of a tumor removed from the pulmonary fields of a woman 56 years old; they believed that this tumor, which incidentally had increased in size on successive examinations, was a hamartoma. In 3 exceptional cases, reported by McDonald and associates,[7] the tumor did not contain cartilage.

There are several interesting features in Case 1. The rapid growth of the tumor over a period of 7 months is unusual. Weisel and co-authors reported 10 hamartomas, all of which had shown evidence of growth on serial films. Most of these had increased in size from a few millimeters to 2.5 cm. over periods of observation of 2 years. In one of the cases cited by Stein and Poppel[6] there was, during a period of 12 years, a slight increase in size which could not be completely accounted for on technical factors. However, most of the reported cases are similar to the 2 case [illegible] followed 5 and 21 years, respectively, by repeated roentg [illegible] nations with no demonstrable increase in size.

to pulmonary parenchyma, the tumor and lung were not separated by a capsule. On cutting through the tumor, the pathologist felt a fine gritty sensation and there were small, white, semitranslucent foci in the medial portion of the tumor. This appeared to be cartilage.

Frozen sections revealed cellular mesenchymal tissue considered to be fibrous or smooth muscle, and the lesion was considered benign, probably representing fibroma or hamartoma of the lung. Microscopic examination of sections from the medial portion of the tumor revealed areas containing hyaline cartilage, fibrous tissue, bronchi, and smooth muscle tissue in a disorderly arrangement. Other sections were composed almost entirely of richly cellular intertwining tissue bundles which, on differential staining, were identified as smooth muscle tissue. It was stated that recent proliferation of the smooth muscle component of the tumor probably accounted for the recent growth observed in the roentgenograms. The tumor was considered to be benign and one lymph node in the specimen showed no neoplastic tissue.

The case was subsequently reviewed by several members of the Armed Forces Institute of Pathology who voiced the unanimous opinion that "the abnormal amount, distribution and morphology of the cartilage component of the lesion frequently related to clefts lined by normal-appearing bronchial epithelium suggested that the lesion was basically a hamartoma. However, the predominating spindle cells, arranged for the most part in interlacing bands, could not be interpreted as simply a component of a hamartoma. The spindle cell growth was believed to represent a true neoplasm, probably of smooth muscle fibers and probably malignant because of the nuclear hyperchromatism and considerable mitotic activity. The lesion as a whole was, therefore, interpreted as a hamartoma in which the smooth muscle component had undergone neoplastic change. The case was coded as lobe of lung, probably leiomyosarcoma arising in hamartoma of lung. The reviewer was not aware of any instance of leiomyoma or leiomyosarcoma arising in this way."

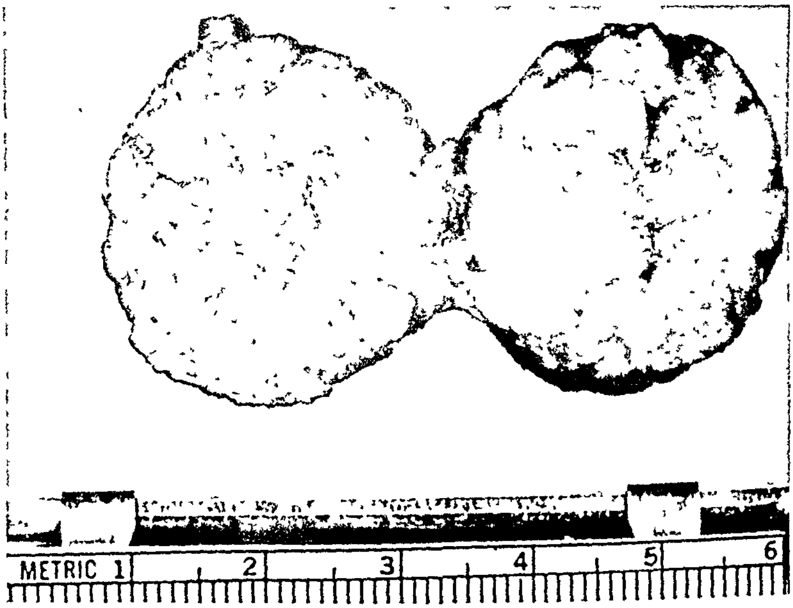

Fig. 3.—Case 3. Gross specimen showing typical appearance of a hamartoma.

Case 2.—A white woman, 27 years old, was found to have a nodule, 2 cm. in diameter, in the right lower pulmonary field adjacent to the subhilar region. The tumor had smooth margins and its density was only slightly greater than that of the surrounding normal pulmonary tissue. The patient was asymptomatic. Results of tuberculin and coccidioidin tests were negative. The small tumor was enucleated with ease from the right upper lobe and the patient's recovery was uneventful.

INEFFECTIVENESS OF ADRENOSEM IN PULMONARY SURGERY

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ADRENOSEM (adrenochrome monosemicarbazone sodium salicylate complex) is a substance related chemically to epinephrine, but it is devoid of sympathomimetic effects. Its use as a hemostatic agent was suggested by the studies of Roskam,[1] who showed that epinephrine favors spontaneous hemostasis after it is oxidized into adrenochrome or a closely related substance. Additional studies by Fulton and co-workers,[2, 3] on the cheek pouch of the hamster, indicated that petechial formation which usually follows tissue trauma is reduced by Adrenosem. It is claimed by the manufacturer that the compound decreases capillary permeability and enhances retraction of severed capillary ends. A number of recent reports concerning the use of the drug to control small vessel bleeding during surgery have been enthusiastic. Peele[4, 5] reported favorable results in tonsil and adenoid operations. He felt that bleeding was diminished by the use of Adrenosem, as evidenced by reduced operating time and less requirement for suture. Cases of epistaxis had decreased need for postnasal packing, and the operative field was better visualized in subglottic surgery. Owings[6] claimed a reduction in postoperative bleeding from 10 per cent to 2 per cent with Adrenosem therapy in nose and throat work. Sherber,[7] Riddle,[8] and Bacala[9] have described their experiences with the drug in a number of surgical procedures, the results of which were all favorable. Some skepticism was expressed, however, in a recent A.M.A. report.[10]

It is difficult to evaluate these reports because of their subjective nature. To date, no studies have been described in which adequate control data were provided. In the past, many alleged hemostatic agents have received brief periods of enthusiasm and were subsequently shown to be ineffective. Perhaps few other areas are so difficult to interpret because of spontaneous variation and lack of strict criteria for effect. The present study was undertaken to attempt an objective evaluation of Adrenosem as a hemostatic agent during surgery.

METHOD

The subjects were patients undergoing thoracic surgery at Montefiore Hospital. In all cases, pneumonectomy or lobectomy was the anticipated operation. The relatively uniform procedure performed by the same operating team appeared to represent a situation most favorable for evaluation of a hemostatic

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The malignant changes present in the tumor in Case 1 as interpreted by members of the Armed Forces Institute of Pathology prompted search of the literature for similar cases. Sherwood and Sherwood[8] reported a fatal case of chondroma of the lung. Simon and Ballon[9] described local cancerous features in a case of hamartoma of the lung.

Also of interest is the young age of the patient in Case 1. In a review of 107 cases of chondromatous hamartomas by Jones,[10] in 1949, the ages ranged from 21 to 78 years. Jones reported a hamartoma, found at necropsy in a newborn infant, which occupied almost the entire upper lobe of the lung.

In none of the 4 cases presented here was calcium demonstrated roentgenographically. Although several authors have emphasized the frequency of calcification in these tumors, it apparently is not as frequent a finding as we have been led to believe. In the 17 cases presented by Lemon and Good,[3] only 3 contained calcium.

There were no roentgenographic features in these 4 cases that would have suggested the diagnosis of hamartoma with a reasonable degree of accuracy. Two of the nodules were opaque to the roentgen rays whereas two were relatively radiolucent with density only slightly greater than that of the surrounding pulmonary tissue. This radiolucency can be ascribed to the high fat content and cystic spaces within the nodular mass. The subpleural location and well-defined borders without reaction of the adjacent pulmonary tissue were features noted that have been previously reported. The latter would assist in excluding tuberculoma, but there are a host of benign lesions and a few malignant ones that have fairly clear-cut borders.

SUMMARY

Four cases of hamartoma of the lung have been presented. One of these tumors, in a baby 22 months old, increased greatly in size over a period of 7 months and microscopic examination revealed malignant changes in the smooth muscle component of the lesion.

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TABLE II. CHANGE IN BLEEDING TIME DURING ONE HOUR INTERVAL BEFORE SURGERY

CHANGE (MIN.)	PLACEBO	ADRENOSEM
+1	1	3
No change	21	20
-1	3	2

TABLE III. TOTAL EVALUATION OF BLEEDING AND OOZING

	MORE	SAME	LESS
Adrenosem	7	6	12
Placebo	6	9	10

DISCUSSION

The data indicate that Adrenosem has no effect in reducing small blood vessel bleeding in pulmonary surgery. Although this type of operation may present special problems, hemostasis is tested in normal skin, muscle, and other tissue. The burden of proof, therefore, is on the shoulders of those who claim that the drug does have benefit. Existing concepts of hemostasis in small blood vessels[11, 12] indicate that the most important single factor involved is the blood platelet. When small vessels are cut, agglutination of the platelets and their adhesion to the damaged vascular wall is the primary event (In those vessels large enough to have a muscular coat, vasoconstriction takes place first.) Then follows the mechanism of blood coagulation, with reinforcement of the platelet plug by a fibrin clot A hemostatic drug, to be effective, should have one or more of the following properties: (a) improve vascular resistance, (b) aid in the formation of the platelet plug, and (c) increase blood coagulability. These effects have not been noted with Adrenosem.

Casual clinical observation cannot be regarded as a reliable method of drug evaluation. Adequately controlled study, preferably using a double blind technique, gives the objective information needed before definite conclusions can be drawn. The situation is reminiscent of periodic discoveries of drugs to treat hemophilia. Such agents as estrogens and ergot alkaloids were stated to be the treatment of choice for this condition but, when results were carefully examined by objective methods, the initial enthusiasm could not be confirmed.[13]

SUMMARY AND CONCLUSIONS

The drug Adrenosem was studied, using a double blind technique. There was no appreciable difference between the effect of the drug and a placebo in the course of its use in pulmonary surgery. We conclude that Adrenosem probably is of no value as a hemostatic agent.

We wish to acknowledge the cooperation of Dr. Elliot Hurwitt, Dr. Morton Rosenblatt, Dr. Aaron Schwartz, and Dr. Stanley Fell, the surgeons whose participation made this study possible.

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agent. Moreover, this type of operation involved considerable bleeding from small vessels. One half received intramuscular Adrenosem,* the other half received injections of a placebo of similar appearance. During the study, the ampules were labeled only by code letters. The key to the code was not available to this group until after the series was completed, and the surgeons did not know which type of injection was given. The two types of ampules were given in a random and predetermined order. Dosage schedules were according to the manufacturer's instructions.

A few days before surgery each patient had a one-stage prothrombin test and a platelet count, performed by phase microscopy. In patients who came to surgery, these were within normal limits. One hour before operation, a Rumpel-Leede test and bleeding time were performed. The Rumpel-Leede test was done with the blood pressure cuff inflated to 90 mm. Hg for exactly 5 minutes. The bleeding time was performed by piercing the ear lobe completely with a No. 11 Bard-Parker blade. At this time 10 mg. (2 ampules) of "medication" were given. Just prior to anesthesia, the Rumpel-Leede test and bleeding time were repeated. Subsequently, intramuscular injection of 5 mg. was given by the anesthetist (a) when the chest was opened, (b) when the operation was deemed half over, as estimated by the surgeon. Additional 5 mg. injections were given 2 and 5 hours postoperatively.

The patients were closely observed for the degree of bleeding, especially small blood vessel oozing. Bleeding from major vessels did not influence the ratings. The postoperative course was evaluated for evidence of further blood loss into the pleural cavity and the operative wound. A final evaluation was made of bleeding during the entire procedure, including the postoperative course, and was classified as more, the same, or less bleeding than expected for that particular type of operation.

TABLE I. RESULTS OF RUMPEL-LEEDE TEST

RESULT	PLACEBO	ADRENOSEM
0	23	21
1+	1	4
2+	1	0
3+	0	0
4+	0	0

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*Provided by the S. E. Massengill Company, Bristol, Tenn.

RESULTS

Table I shows the result of the Rumpel-Leede test performed one hour before surgery, at which time the first dose of "medication" was given. The same test was repeated immediately before surgery. In no cases was there a change. Table II indicates that there were only minor changes in bleeding time in the one-hour interval. Total evaluation of the results is shown in Table III. It appears that there is no demonstrable difference in the amount of bleeding and oozing when a placebo is used in place of Adrenosem.

THE AMNESIC EFFECT OF PROMETHAZINE HYDROCHLORIDE IN BRONCHOSCOPY

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AMNESIA is a desirable phenomenon in the patient undergoing bronchoscopic examination. Bronchoscopy under topical anesthesia in the conscious or alert patient is an unpleasant experience for both patient and endoscopist. For adequate examination of patients who cannot cooperate, bronchoscopies are frequently performed under general anesthesia. However, the bronchoscope cannot be introduced in the somnolent patient unless the jaw muscles are relaxed.

Relaxation can be achieved by use of general anesthesia, by other central nervous system depressants, or by a muscle relaxant such as succinylcholine chloride. However, these techniques tend to produce respiratory depression. Hypoxemia can result, with all its grave consequences. To avoid hypoxia and hypoxemia, a portable chest respirator has been employed in conjunction with anesthesia for bronchoscopy.[1]

In our opinion, general anesthesia or use of the chest respirator plus topical anesthesia introduce unnecessarily complicating factors into bronchoscopy. Such procedures inevitably involve more risk to the patient than actual insertion of the bronchoscope.

Ideally, bronchoscopy calls for: (1) an awake, cooperative patient who can respond to and obey requests of the operator; (2) a relaxed and comfortable patient with a sense of well-being; (3) a patient without respiratory depression; and (4) one who later lacks unpleasant memory of the bronchoscopic procedure.

Anderson[2] performed 290 bronchoscopies with cocaine hydrochloride topical anesthesia and intravenous meperidine hydrochloride as a sedative-analgesic. His patients were reportedly comfortable and relaxed, although awake and cooperative. Some cases were amnesic.

Altman and Fialkov[3] reported 156 bronchoscopies performed with tetracaine hydrochloride-cocaine hydrochloride topical anesthesia and intravenous meperidine hydrochloride as a sedative-analgesic. No anxiety was noted during bronchoscopy and amnesia was observed in a few patients.

The use of promethazine hydrochloride (Phenergan hydrochloride) in bronchoscopic examination has not been reported, yet the drug would appear to hold merit for such procedures. Sadove,[4] Weiss and McGee,[5] and Holzmann and Josipowicz[6] described the sedative and antiemetic action of promethazine, and its value in anesthetic techniques. None of these investigators emphasized the amnesic effect of promethazine, although Weiss reported its existence in older patients.

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RESULTS

Meperidine and Hexylcaine.—No patient experienced complete amnesia during examination. The typical patient partially recalled the procedure and remembered periods of stimulation by the bronchoscope. No patient felt the experience was unpleasant and all expressed willingness to undergo bronchoscopy again if necessary. All subjects were awake and oriented at the end of the procedure. They were returned to the wards immediately and needed little postoperative care. Only 2 of 25 patients attained the desired stage of relaxation on 100 mg. of meperidine or less. No laryngospasm was observed. Table I lists other pertinent data and tabulates results.

TABLE I. COMPARATIVE AMNESIC RESPONSE TO BRONCHOSCOPY FOLLOWING ANESTHESIA

ANESTHETIC TECHNIQUE	INTRAVENOUS MEPERIDINE HCl PLUS TOPICAL HEXYLCAINE HCl	INTRAVENOUS PROMETHAZINE HCl AND MEPERIDINE HCl PLUS TOPICAL HEXYLCAINE HCl
Number of patients	25	25
Age range, years	17-71	17-80
Median age, years	58	39
Range of meperidine HCl dose, mg.	90-200	40-200
Mean meperidine HCl dose, mg.	170	130
Promethazine HCl dose, mg.	—	50
Response		
Complete amnesia	0	15
Partial amnesia	1	6
No amnesia	24	4
	25	25

Promethazine, Meperidine, and Hexylcaine.—Of 25 patients, 15 had complete amnesia during bronchoscopy. Six vaguely recalled momentary and not unpleasant stimulation at some time during the procedure. Of those who remembered examination, none found it unpleasant. All were cooperative and obeyed commands when given. All patients were returned to their wards shortly
fter the procedure. Patients were awake or easily aroused and needed little
stoperative care. Ten of 25 patients required 100 mg. of meperidine or less
adequate response. No laryngospasm was observed. Table I lists results.

With 3 patients, meperidine induction was intended. They became tense
ncooperative during an early stage of induction, and so 50 mg. of prometh-
was given intravenously. All 3 then relaxed; induction was completed
procedure was carried out without incident and with complete amnesia.
ases were tabulated as partially amnesic since memory was retained
tempted meperidine induction.

FIG patients experienced amnesia that began only 2 or 3 min
ine injection. In some cases the amnesic effect extended w
rative period. Several patients commented they had "slept
intra fter bronchoscopy although in fact they had been easily aro
findin d to commands during this period.

trache patients underwent two bronchoscopies, one with promethazine an
is obvi drug. Both experienced complete amnesia when promethazine
iner att

Rece

PLAN OF STUDY

Bronchoscopic examination was performed on 50 consecutive, unselected
patients where the procedure was indicated. Twenty-five patients received intra-
venous meperidine hydrochloride for sedation and analgesia, and hexylcain
hydrochloride for topical anesthesia. The remaining 25 cases received intr
venous promethazine hydrochloride* in addition to meperidine and hexylcain

Method.—In general, preoperative medication consisted of: (1) pen
barbital or Secobarbital, 90 mg., by mouth 90 minutes prior to bronchoscopy,
(2) 60 mg. codeine hydrochloride and 0.43 mg. atropine sulfate in a si
intramuscular injection 60 minutes before bronchoscopy.

At time of examination, the patient lay in supine position on the bron
copy table. An intravenous drip of 5 per cent dextrose in water was st
A three-way stopcock in the tubing facilitated control of the intravenous
injections. Blood pressure, pulse rate, respiratory rate, and depth were
tored throughout examination.

Among those receiving promethazine, induction was begun with
0 mg. intravenous injection of the drug. Immediately thereafter,
meperidine was introduced through the tubing. The patient was th
tioned and observed for relaxation and a "feeling of lightheadedne
meperidine dose was repeated in 20 mg. increments at 2-minute inte
the patient appeared to sleep lightly.

In the "dozing" stage, patients who received promethazine re
commands, were cooperative, and were easily aroused. To reach t
sedation, the mean meperidine dose was 130 mg. and ranged from 4
At least 15 minutes were required for peak effect of the meperi

When the patient appeared relaxed, the posterior pharynx an
sprayed with a 5 per cent aqueous solution of hexylcaine hydroch
anesthetization of the vocal cords, 2 c.c. of 5 per cent hexylcain
injected between them. A syringe and flexible laryngeal cannula
last operation which served to anesthetize the tracheobronchial t
copy, was then undertaken.

The 25 cases not receiving promethazine were treated in i
save that induction started with introduction of 20 mg. mep
these patients, the "dozing" state could not be attained with
dosage that would also bring about unwanted respiratory
bronchoscopy in these patients began when the patient wa
relaxed after a mean meperidine dose of 150 mg.

The sedative and anesthetic procedures involved an av
in all 50 patients. Amnesia or its lack was ascertained by
questioning of each patient postoperatively. A standard
put which traced the patient's memory of events minute
time of leaving the ward. Complete amnesia was record
had no memory whatever of the procedure.

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*Promethazine hydrochloride (N-[2′-dimethylamino-2′-methyll
was supplied for this study by Wyeth Laboratories,
ydrochloride.

RIGHT-LEFT IDENTIFICATION OF BRONCHOSCOPY SMEARS

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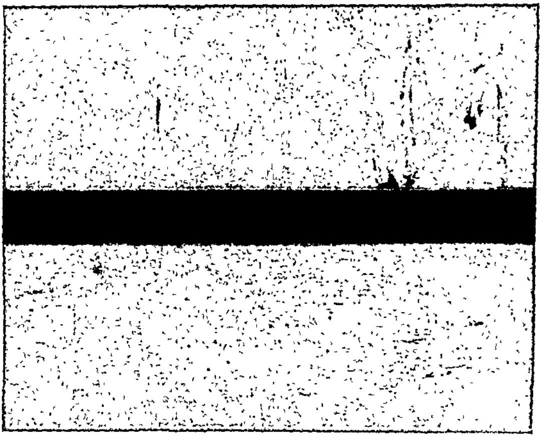
BRONCHIAL secretions, like respiratory air, share the upper respiratory tract to the right and left bronchi. Since this common passageway is both an entrance and an exit, there exists ample opportunity for mixing secretions on expiration and contralateral inoculation on inspiration. It is therefore impossible for the bronchoscopist to collect a smeared or aspirated specimen which represents a single side exclusively. Regardless of this limitation, determination of a preponderant bacterium or type of desquamated cell on a single side may aid greatly in diagnosis.



ig. 1.—*Top*, Papanicolaou smear from right side. *Bottom*, Acid-fast smear from left side.

When nonlocalizing but symptomatic conditions such as hemoptysis or ntractable cough occur in the absence of positive physical or roentgenographic ndings, a bronchoscopic examination should be done. The portion of the acheobronchial tree accessible to visual and "instrumentation-tactile" sense obviously limited. When no lesion is encountered in that portion, the examner attempts to increase his area of study by collecting smeared and aspirated

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vas employed and no amnesia when the drug was not used. Of these 2 patients disliked the amnesic sensation under promethazine-meperidine anesthesia and preferred to retain his memory of events.

A careful watch was maintained for untoward response, particularly any change in blood pressure, pulse rate, and respiratory rate. No significant difference was observed in these physiologic phenomena and no side effects were noted.

COMMENT

Discomfort and pain associated with bronchoscopy accomplished solely under topical anesthesia leaves the patient with an unpleasant memory. Also, under such conditions, an adequate examination is not always possible.

Promethazine affords the patient amnesia and obliterates the memory of an unpleasant endoscopic procedure. Meperidine fortified with promethazine is an even more desirable drug regimen in that the sedative-analgesic combination provides a sense of well-being undisturbed by pressure or position discomfort. This procedure avoids use of general anesthesia and the attendant risks to obtain desired cooperation and relaxation. Table I also suggests promethazine permits use of less analgesic than would otherwise be required to carry ou bronchoscopy without discomfort.

Our experience leads us to the opinion that amnesia is most desirable ing bronchoscopy. Hence, we consider promethazine essential to the preparation of the patient for such procedures. The technique describ would appear suitable for wider usage, although local circumstances slight modifications.

INDEX TO VOLUME 35

A

Adkins, Paul C (with Newman, William), Three primary carcinomas of lung arising in left lower lobe with metastasis of two of the tumors, 474
Adrenosem in pulmonary surgery, ineffectiveness of (Marcus and Spaet), 821
Allen, George T (with Miller, R Drew, Bridge, Ezra V, Jr, Fowler, Ward S, Helmholz, H Frederic, Jr, and Ellis, F Henry, Jr), Pulmonary function before and after pulmonary resection in tuberculous patients, 651
Alley, R D (with Lueders, H W, Stranahan, Allan, Kausel, H W, and Peck, A S), Simplified method for achieving intrarespiratory humidification in tracheotomized patient, 461
American Association for Thoracic Surgery, Program 38th Annual Meeting, 555
Analgesia following major thoracic surgery, morphine and amiphenazole for postoperative (Weber), 105
Andersen, Howard A (with Bernatz Philip E, Smith, J Lawrence, and Ellis, F Henry, Jr), Benign, pedunculated, intraluminal tumors of esophagus, 503
Andreae, Eric (with Shimomura Seiichi, Bell, A L Loomis, Jr, and Fitzpatrick, Hugh F), Pressure flow relationships of Sigmamotor pump as used in cardiac bypass, 747
Angiocardiography as aid to identification of nonresectable pulmonary carcinomas Wyman and Wilkins), 452
Antonius, N A (with Creech, A D, and Massarelli, L G), Clinical evaluation of surgical treatment of thirty two cases of coronary artery disease, 68
Aorta, coarctation of, with a report of unusual operative complication (Knuth, Walske, and Neis), 591
  new classification for coarctations of (Lemmon and Bailey), 291
  transfixion of, accompanied by a Brown Séquard syndrome (Baret, DeLong, Tukanowicz, and Blakemore), 359
Aortic insufficiency by circumclusion, surgical correction of (Taylor, Thrower, Black, and Harken), 192
  stenosis, congenital, during hypothermia, visual repair of (Swan, Wilkinson and Blount), 139
  valve without occlusion or bypass, direct vision approach to (Gadboys, Harrison, and Litwak), 756
Arany, L S (with White, Manuel), Resection in pulmonary cryptococcosis (torulosis), 402
Arteries, coarctation of aorta proximal to both subclavian (Brynolf, Crafoord, and Mannheimer), 123
Ashe, William M (with Hutcheson, James B, and Paulson, Donald L), Lipomas of bronchus, 638
Askari, Salah Al (with Ford, Joseph M, Clauss, Roy H, and Wylie, Robert H), Surgical treatment of pulmonary hemorrhage in tuberculosis, 341
Atresia, unusual esophageal, with distant membranous obstruction of esophagus (Overton and Creech), 674
Atrioseptopexy simplified new technique (Hirose, Morse, and Bailey), 255
Auerbach, S H (with Matuska, R A, Hahn, P F, Carlson, R I, and Meneely, G R), Further observations on lymphatic pick up of radioactive silver coated gold colloid administered intrathoracically to dogs, 135
Aufses, Arthur H (with Harte, Maurice S), Extrapleural thoracoplasty in treatment of pulmonary tuberculosis an eight to twenty one year follow up study, 332
Aves, Fred H (with Leidler, Franz, and McKnight, Evelyn), Right left identification of bronchoscopy smears, 829

B

Badeer, Henry, Ventricular fibrillation in hypothermia a review of factors favoring fibrillation in hypothermia with and without cardiac surgery, 265
Bailey, Charles P, Discussion of papers on correction of aortic insufficiency and surgical correction of mitral insufficiency, 231
  discussion of papers on repair of aortic stenosis during hypothermia and surgical treatment of valvular lesions, 190
  discussion of ventriculoplasty for cardiac aneurysm, 67
  (with Bolton, Houck, Nichols, Henry, and Gilman, Richard A), Ventriculoplasty for cardiac aneurysm, 37
  (with Hirose Teruo), Maximal reconstitution of stenotic mitral valve by neostrophingic mobilization (rehinging of se [illegible] aflet), 559

specimens for cytologic and bacteriologic examinations. These specimens represent material coughed into the main-stem bronchi from the more peripheral bronchioles.

In the darkened room used for bronchoscopic examination it is sometimes difficult to identify adequately by marking pencils, gummed labels, etc., smear preparations collected from the right and left sides. A very simple means of identification which places the responsibility entirely upon the bronchoscopist, and has been successfully used at the Memorial Hospital is:

> *R*ight side smears are smeared at *R*ight angles to the long axis of the glass slide.
>
> *L*eft side smears are swabbed *L*engthwise or in the long axis of the glass slide.

This method works equally well for Papanicolaou and acid-fast smears as is demonstrated in Fig. 1. By holding the slide up to a light source, the pathologist can easily and grossly identify the side from which that particular smear was prepared.

- Brofman, B. L. (with Beck, Claude S.), Coronary heart disease. I. Hemodynamic principles and their therapeutic applications, 232, 254
  - (with Charms, B. L., Elder, J. C., and Kohn, P. M.), Unilateral pulmonary artery occlusion in man. II. Studies in patients with chronic pulmonary disease, 316
- Bronchial adenoma treated by sleeve resection of main stem bronchus (Connolly and Chamberlain), 372
  - closure with catgut sutures (Sherman and Conant), 363
  - polyps, solitary, of inflammatory origin (Salek, Pazderka, and Žak), 807
  - stenosis, kinked bronchus simulating (Bjork), 368
- Bronchogenic cysts and theory of intralobar sequestration: new embryologic data (Boyden), 604
- Bronchographic evaluation of treatment by internal suture of the pericyst, hydatid cyst of the lung with (Srouji, Mullim, and Wilson), 779
- Bronchoscopy, amnesic effect of promethazine hydrochloride in (Pino and Van Houten), 825
  - smears, right-left identification of (Aves, Leidler, and McKnight), 829
- Bronchus, kinked, bronchial stenosis simulating (Bjork), 368
  - lipomas of (Hutcheson, Ashe, and Paulson), 638
  - mucormycosis of, successfully treated by lobectomy (Dillon, Sealy, and Fetter), 464
- Brown, Robert B. (with Peabody, J. Winthrop, Jr., Sullivan, M. Bruce, and Cannon, Albert), Mediastinal granulomas: revised concept of their incidence and etiology, 384
- Brown-Sequard syndrome, transfixion of aorta accompanied by (Baret, DeLong, Tukanowicz, and Blakemore), 359
- Brynolf, I. (with Crafoord, C., and Mannheimer, E.), Coarctation of aorta proximal to both subclavian arteries, 123
- Bypass, direct vision approach to aortic valve without occlusion or (Gadboys, Harrison, and Litwak), 756

C

- Cannon, Albert (with Peabody, J. Winthrop, Jr., Brown, Robert B., and Sullivan, M. Bruce), Mediastinal granulomas: revised concept of their incidence and etiology, 384
- Capillary telangiectasia (Rendu-Osler-Weber disease), pulmonary arteriovenous fistula associated with: report of case illustrating use of metal casting for demonstrating lesion (Steinberg, Maisel, and Vogel), 517
- Carcinoma in bronchial stump, radiation therapy for recurrent (Pinsky and Emerson), 683
  - of lung treated by lobectomy: problem of local recurrence (Holland), 274
- Carcinomas, three primary, of lung arising in left lower lobe with metastasis of two of the tumors (Newman and Adkins), 474
- Cardiac aneurysm, ventriculoplasty for (Bailey, Bolton, Nichols, and Gilman), 37
  - bypass, pressure-flow relationships of Sigmamotor pump as used in (Shimomura, Andrew, Bell, and Fitzpatrick), 747
  - surgery, bacterial and mycotic endocarditis following (Hyun, Dawson, and Pence), 298
- Cardiopulmonary bypass, bubble diffusion oxygenator for (Cooley, Belmonte, Latson, and Pierce), 131
- Cardiovascular structures, resistance to catheter puncture of (Grajo and Lurie), 751
- Carlson, R. I. (with Matuska, R. A., Hahn, P. F., Auerbach, S. H., and Meneely, G. R.), Further observations on lymphatic pick-up of radioactive silver-coated gold colloid administered intrathoracically to dogs, 135
- Carotid arteries implanted into myocardium, maintenance of patency of (Nachlas, Myers, Solomon, Kelly, and Seligman), 706
- Catgut sutures, bronchial closure with (Sherman and Conant), 363
- Catheter puncture of various cardiovascular structures, resistance to (Grajo and Lurie), 751
- Cavin, Elwyn (with Masters, Joseph H., and Moody, James), Hamartoma of lung, 816
- Chamberlain, J. Maxwell (with Connolly, John E.), Bronchial adenoma treated by sleeve resection of main stem bronchus, 372
- Charms, B. L. (with Brofman, B. L., Elder, J. C., and Kohn, P. M.), Unilateral pulmonary artery occlusion in man. II. Studies in patients with chronic pulmonary disease, 316
- Charnas, Raymond M. (with Bergmann, Martin), Tracheobronchial rests in esophagus: relation to some benign strictures and certain types of cancer of esophagus, 97
- Chesney, John (with Fomon, John J., Davis, Harold, Kurzweg, Frank T., and Broadaway, Rufus K.), Single dosage x-ray radiation to lung and posterior mediastinum through open chest. I. Results in dogs receiving dosage between 6,000 and 15,000 r, 771

Bailey, Charles P.—Cont'd
  (with Hirose, Teruo, and Morse, Dryden P.), Atrioseptopexy: simplified new technique, 255
  (with Lemmon, W. M.), New classification for coarctations of aorta, 291
  (with Musser, Benjamin G.), Fenestrated cardiovascular clamps, 519
Bakst, Alvin A. (with Boley, Scott J., Joseph, Edward, and Loewe, Leo), Surgical correction of mitral insufficiency by use of pericardial valve graft, 492
Baret, Alexander C. (with DeLong, Robert P., Tukanowicz, S. Anthony, and Blakemore, William S.), Transfixion of aorta accompanied by a Brown-Séquard syndrome, 359
Bauer, Albert J (with Sweetman, William R., Hartley, Lawrence J., and Salyer, John M.), Postinflammatory "tumor" of lung, 802
Beattie, E. J., Jr. (with Sanborn, E. B., and Slaughter, D. P.), Secondary neoplasms of mediastinum, 678
Beck, Claude S., Discussion of ventriculoplasty for cardiac aneurysm, 67
  (with Brofman, B. L.), Coronary heart disease. I. Hemodynamic principles and their therapeutic applications, 232
Bell, A. L. Loomis, Jr. (with Shimomura, Seiichi, Andreae, Eric, and Fitzpatrick, Hugh F.), Pressure-flow relationships of Sigmamotor pump as used in cardiac bypass, 747
Belmonte, Benjamin A. (with Cooley, Denton A., Latson, Joseph R., and Pierce, James F.), Bubble diffusion oxygenator for cardiopulmonary bypass, 131
Bercu, Bernard A. (with Diettert, Gerald A., and Ferguson, Thomas B.), Use of modified Gibbon-type pump-oxygenator. I. Description of apparatus, 416
Beretta, Francesco F. (with Jordan, Prescott, Jr., and Tolstedt, Grandon E.), Stainless steel bubble oxygenator, 411
Bergman, Frank (with Linder, Erland), Tumor-forming amyloidosis of lung, 628
Bergmann, Martin (with Charnas, Raymond M.), Tracheobronchial rests in esophagus; relation to some benign strictures and certain types of cancer of esophagus, 97
Berman, Edward J. (with Taylor, Max T., and Fisch, Charles), Experimental prevention of ventricular fibrillation following hypothermia and induced cardiac arrest, 483
Bernatz, Philip E. (with Smith, J. Lawrence, Ellis, F. Henry, Jr., and Andersen, Howard A.), Benign, pedunculated, intraluminal tumors of esophagus, 503
Bewick, John (with Fowler, Noble O., and Couves, Cecil), Effect of inflow obstruction and rapid bleeding on ventricular diastolic pressure, 532
Bishof, Josephine Kain (with Truex, Raymond C.), Conduction system in human hearts with interventricular septal defects, 421
Björk, Viking Olov, Kinked bronchus simulating bronchial stenosis, 368
  (with Rodriguez, Louis E.), Reconstruction of trachea and its bifurcation, 596
Black, Harrison (with Taylor, Warren J., Thrower, Wendell B., and Harken, Dwight E.), Surgical correction of aortic insufficiency by circumclusion, 192
Blades, Brian (with Magovern, George J.), Primary extragenital chorioepithelioma in male mediastinum, 378
Blakemore, William S. (with Baret, Alexander C., DeLong, Robert P., Tukanowicz, S. Anthony), Transfixion of aorta accompanied by a Brown-Séquard syndrome, 359
Blount, S. Gilbert, Jr. (with Swan, Henry, and Wilkinson, Robert H.), Visual repair of congenital aortic stenosis during hypothermia, 139
Boley, Scott J. (with Bakst, Alvin A., Joseph, Edward, and Loewe, Leo), Surgical correction of mitral insufficiency by use of pericardial valve graft, 492
Bolton, Houck E. (with Bailey, Charles P., Nichols, Henry, and Gilman, Richard A.), Ventriculoplasty for cardiac aneurysm, 37
Bosher, Lewis H., Jr., Discussion of ventriculoplasty for cardiac aneurysm, 65
  (with Meredith, J. M., and Kay, Saul), Case of granular cell myoblastoma (organoid type) involving arm, lung, and brain, with twenty years' survival, 80
Boyden, Edward A., Bronchogenic cysts and theory of intralobar sequestration: new embryologic data, 604
Brantigan, O. C. (with de Borja, P. S.), Simple method of scapular traction for closure of lateral or posterolateral chest incisions, 400
Bridge, Ezra V., Jr. (with Miller, R. Drew, Fowler, Ward S., Helmholz, H. Frederic, Jr., Ellis, F. Henry, Jr., and Allen, George T.), Pulmonary function before and after pulmonary resection in tuberculous patients, 651
Broadaway, Rufus K. (with Fomon, John J., Davis, Harold, Kurzweg, Frank T., and Chesney, John), Single dosage x-ray radiation to lung and posterior mediastinum through open chest. I. Results in dogs receiving dosage between 6,000 and 15,000 r, 771

DeWall, Richard A (with Lillehei, C Walton Gott, Vincent L, and Varco, Richard L), Surgical treatment of stenotic or regurgitant lesions of mitral and aortic valves by direct vision utilizing pump oxygenator, 154

Diettert, Gerald A (with Bercu, Bernard A, and Ferguson, Thomas B), Use of modified Gibbon type pump oxygenator I Description of apparatus, 416

Dillard, Richard A (with Perkins, Rex B), Spontaneous bilateral chylopneumothorax, 91

Dillon, M L (with Sealy, W C, and Fetter, B F), Mucormycosis of bronchus successfully treated by lobectomy, 464

Duchesne, Edward R (with Fowler, Noble O), Effect of experimental pulmonary valvular insufficiency on circulation, 643

E

Ectopia cordis, partial surgical management of congenital bifid sternum with (Sabiston), 118

Edwards, Jesse E (with Gladding Thomas C, and Weir, Alva B, Jr), Congenital communication between right coronary artery and right atrium 662

Eiseman, B (with Rainer, W G), Clinical management of posttraumatic rupture of thoracic aorta, 347

Elder, J C (with Charms, B L, Elder, J C, and Kohn, P M), Unilateral pulmonary artery occlusion in man II Studies in patients with chronic pulmonary disease, 316

Ellis, F Henry, Jr, Discussion of papers on correction of aortic insufficiency and surgical correction of mitral insufficiency, 230
  (with Bernatz, Philip E Smith, J Lawrence, and Andersen, Howard A), Benign, pedunculated, intraluminal tumors of esophagus 503
  (with Miller, R Drew, Bridge, Ezra V, Jr, Fowler, Ward, S, Helmholz H Frederic, Jr, and Allen, George T), Pulmonary function before and after pulmonary resection in tuberculous patients, 651

Emerson, George L (with Pinsky, Harry J), Radiation therapy for recurrent carcinoma in bronchial stump, 683

Endocarditis, bacterial and mycotic, following cardiac surgery (Hyun, Dawson, and Pence), 298

Esophagus, benign, pedunculated, intraluminal tumors of (Bernatz, Smith, Ellis, and Andersen), 503

Esophagus—Cont'd
  reconstruction of, with segments of colon (Neville and Clowes), 2
  tracheobronchial rests in (Bergmann and Charnas), 97
  unusual esophageal atresia with distant membranous obstruction of (Overton and Creech), 674

Experimental bilateral transsternal thoracotomy factors improving survival (Kortz), 305

F

Fatti, L Discussion of primary tumors of heart, 35

Fell, Egbert H, Discussion of primary tumors of heart, 33
  Discussion of ventriculoplasty for cardiac aneurysm, 64

Fenestrated cardiovascular clamps (Bailey and Musser), 549

Ferguson, Thomas B (with Diettert, Gerald A, and Bercu, Bernard A), Use of modified Gibbon type pump oxygenator I Description of apparatus, 416

Fetter, B F (with Dillon M L, and Sealy, W C), Mucormycosis of bronchus successfully treated by lobectomy, 464

Fisch, Charles (with Berman Edward J, and Taylor, Max T), Experimental prevention of ventricular fibrillation following hypothermia and induced cardiac arrest, 483

Fistula, surgical repair of traumatic aortic right ventricular (King and Shumacker), 734

Fitzpatrick, Hugh F (with Shimomura, Seiichi, Andreae, Eric, and Bell, A L Loomis, Jr), Pressure flow relationships of Sigmamotor pump as used in cardiac bypass, 747

Fomon, John J (with Davis, Harold, Kurzweg, Frank T, Broadway, Rufus K, and Chesney, John), Single dosage x ray radiation to lung and posterior mediastinum through open chest I Results in dogs receiving dosage between 6,000 and 15,000 r, 771

Ford, Joseph M (with Clauss, Roy H, Askari, Salah M, and Wylie, Robert H), Surgical treatment of pulmonary hemorrhage in tuberculosis, 341

Foti, Egidio (with Robicsek, Francis, Magistro, Roberto, Robicsek, Livia, and Sanger, Paul W), Vena cava—pulmonary artery anastomosis for vascularization of lung, 440

Foushee, J H Smith (with Norris, Franklin G), Pulmonary aspergillosis, case report, 542

Chest incisions, simple method of scapular traction for closure of lateral or posterolateral (Brantigan and de Borja), 400
Choy, S. H. (with Hennigar, Gordon R.), Accessory lung with persistent left superior vena cava and duplication of intestine, 469
Churchill, Edward D., Wound surgery encounters a dilemma, 279
Chylopneumothorax, spontaneous bilateral (Dillard and Perkins), 91
Clamp, lung exclusion: new instrument for wedge resections (Hudson), 553
Clamps, fenestrated cardiovascular (Bailey and Musser), 549
Clauss, Roy H. (with Ford, Joseph M., Askari, Salah Al, and Wylie, Robert H.), Surgical treatment of pulmonary hemorrhage in tuberculosis, 341
  (with Wilson, David W.), Pancreatic pseudocyst of mediastinum, 795
Clift, John V. (with Patt, Howard H., Golberg, Mauricio, and Lourvanij, Bunket), Evaluation of implantation of internal mammary artery into myocardium of pigs and dogs, 699
Clowes, George H. A., Discussion of papers on repair of aortic stenosis during hypothermia and surgical treatment of valvular lesions, 190
  (with Neville, William E.), Reconstruction of esophagus with segments of colon, 2
Coarctation of aorta with report of unusual operative complication (Knuth, Walske, and Neis), 591
  proximal to both subclavian arteries (Brynolf, Crafoord, and Mannheimer), 123
Coarctations of aorta, a new classification for (Lemmon and Bailey), 291
Colon, reconstruction of esophagus with segments of (Neville and Clowes), 2
Conant, James S. (with Sherman, Paul H.), Bronchial closure with catgut sutures, 363
Congenital bifid sternum with partial ectopia cordis, surgical management of (Sabiston), 118
  communication between right coronary artery and right atrium (Edwards, Gladding, and Weir), 662
  heart disease, intracardiac pressure measurement during operation for (Lundberg), 740
Connolly, John E. (with Chamberlain, J. Maxwell), Bronchial adenoma treated by sleeve resection of main-stem bronchus, 372
Cooley, Denton A., Discussion of ventriculoplasty for cardiac aneurysm, 66
  Discussion of papers on repair of aortic stenosis during hypothermia and surgical treatment of valvular lesions, 190
Cooley, Denton A.—Cont'd
  (with Belmonte, Benjamin A., Latson, Joseph R., and Pierce, James F.), Bubble diffusion oxygenator for cardiopulmonary bypass, 131
Coronary artery disease, clinical evaluation of surgical treatment of thirty-two cases (Antonius, Crecca, and Massarelli), 68
    occlusion, Ivalon graft to bridge (Smith and McIntyre), 513
    right, congenital communication between right atrium and (Edwards, Gladding, and Weir), 662
  heart disease. I. Hemodynamic principles and their therapeutic applications (Brofman and Beck), 232
Couves, Cecil (with Fowler, Noble O., and Bewick, John), Effect of inflow obstruction and rapid bleeding on ventricular diastolic pressure, 532
Crafoord, C. (with Brynolf, I., and Mannheimer, E.), Coarctation of aorta proximal to both subclavian arteries, 123
Crecca, A. D. (with Antonius, N. A., and Massarelli, L. G.), Clinical evaluation of surgical treatment of thirty-two cases of coronary artery disease, 68
Creech, Oscar (with Overton, Robert C.), Unusual esophageal atresia with distant membranous obstruction of esophagus, 674

D

Daniel, Rollin A., Jr., Surgical treatment of pectus excavatum, 719
Davila, Julio C. (with Glover, Robert P., Voci, Gerardo, Jumbala, P., Trout, Robert G., and Fritz, A. Jane), Clinical and physiologic criteria for surgical correction of mitral insufficiency 206, 231
Davis, Harold (with Fomon, John J., Kurzweg, Frank T., Broadaway, Rufus K., and Chesney, John), Single dosage x-ray radiation to lung and posterior mediastinum through open chest. I. Results in dogs receiving dosage between 6,000 and 15,000 r, 771
Dawson, Elizabeth A. (with Hyun, Bong Hak, and Pence, Robert E.), Bacterial and mycotic endocarditis following cardiac surgery, 298
De Borja, P. S. (with Brantigan, O. C.), Simple method of scapular traction for closure of lateral or posterolateral chest incisions, 400
DeLong, Robert P. (with Baret, Alexander C., Tukanowicz, S. Anthony, and Blakemore, William S.), Transfixion of aorta accompanied by a Brown-Séquard syndrome, 359

Herron, Paul W, Discussion of papers on repair of aortic stenosis during hypothermia and surgical treatment of valvular lesions, 190

Hirose, Teruo (with Bailey, Charles P), Maximal reconstitution of stenotic mitral valve by neostrophingic mobilization (rehinging of septal leaflet), 559
  (with Morse, Dryden P, and Bailey, Charles P), Atrioseptopexy simplified new technique, 255

Holland, Robert H, Carcinoma of lung treated by lobectomy problem of local recurrence, 274

Hopkins, William A, Discussion of primary tumors of heart, 33

Hudson, Theodore R, Lung exclusion clamp new instrument for wedge resections, 553

Hutcheson, James B (with Ashe, William M, and Paulson, Donald L), Lipomas of bronchus, 638

Hydatid cyst of lung with bronchographic evaluation of treatment by internal suture of pericyst (Srouji, Mullim, and Wilson), 779

Hypothermia and induced cardiac arrest, experimental prevention of ventricular fibrillation following (Berman, Taylor, and Fisch), 483
  ventricular fibrillation in (Badeer), 265
  visual repair of congenital aortic stenosis during (Swan, Wilkinson, and Blount), 139

Hyun, Bong Hak (with Dawson, Elizabeth A, and Pence, Robert E), Bacterial and mycotic endocarditis following cardiac surgery, 298

I

Internal mammary artery, implantation of, into myocardium of pigs and dogs, evaluation of (Patt, Golberg, Clift, and Lourvanij), 699
  venography (Schobinger), 692

Intracardiac pressure measurement during operation for congenital heart disease (Lundberg), 740

Intralobar sequestration, bronchogenic cysts and theory of new embryologic data (Boyden), 604

Intrarespiratory humidification in tracheotomized patient, simplified method for achieving (Lueders Stranahan, Alley, Kausel, and Peck), 461

Ivalon graft to bridge coronary artery occlusion (Smith and McIntyre), 513

J

Jarvis, Fred, Discussion of reconstruction of esophagus with segments of colon, 19

Johnston, J Harvey Jr (with Webb Watts R, and Geisler James W), Pneumomediastinum physiologic observations, 309

Jones, John C, Discussion of ventriculoplasty for cardiac aneurysm 66

Jordan, Prescott, Jr (with Holstedt Franklin E, and Beretta, Francis F) Stainless steel bubble oxygenator 411

Joseph, Edward (with Bakst, Alvin A B[illegible] Scott J, and Loewe, Leo), Surgical correction of mitral insufficiency by use of pericardial valve graft, 49

Jumbala, P (with Davila, Julio C, Glover Robert P, Voci, Gerardo, Trout Robert G, and Fritz, A Jane), Clinical and physiologic criteria for surgical correction of mitral insufficiency, 206

K

Karlson, Karl E (with Timmes, Joseph J), Granulomata of mediastinum surgically treated and followed up to nine years, 617

Kasserman, H, Pericardial fat necrosis an unusual entity, 689

Kausel, H W (with Lueders, H W, Stranahan, Allan, Alley, R D, and Peck, A S), Simplified method for achieving intrarespiratory humidification in tracheotomized patient, 461

Kay, Saul (with Meredith, J M, and Bosher, Lewis H, Jr), Case of granular cell myoblastoma (organoid type) involving arm, lung, and brain, with twenty years' survival, 80

Kelly, Alma B (with Nachlas, Marvin M, Myers, Myron J, Solomon, Robert D, and Seligman, Arnold M), Maintenance of patency of carotid arteries implanted into myocardium, 706

King, Harold (with Shumacker, Harris B, Jr), Surgical repair of traumatic aortic right ventricular fistula, 734

Kirklin, John W (with McGoon, Dwight C), Surgical technique for repair of high ventricular septal defects, 581

Knuth, Warren P (with Walske, Benedict R, and Ness, Delbert D), Coarctation of aorta with report of unusual operative complication, 591

Kohn, P M (with Charms, B L, Brofman, B L, and Elder, J C), Unilateral pulmonary artery occlusion in man II Studies in patients with chronic pulmonary disease, 316

Kortz, Allan B, Exxperimental bilateral transsternal thoracotomy factors improving survival, 305

Fowler, Noble O. (with Couves, Cecil, and Bewick, John), Effect of inflow obstruction and rapid bleeding on ventricular diastolic pressure, 532
(with Duchesne, Edward R.), Effect of experimental pulmonary valvular insufficiency on circulation, 643

Fowler, Ward S. (with Miller, R. Drew, Bridge, Ezra V., Jr., Helmholz, H. Frederic, Jr., Ellis, F. Henry, Jr., and Allen, George T.), Pulmonary function before and after pulmonary resection in tuberculous patients, 651

Fritz, A. Jane (with Davila, Julio C., Glover, Robert P., Voci, Gerardo, Jumbala, P., and Trout, Robert G.), Clinical and physiologic criteria for surgical correction of mitral insufficiency, 206

G

Gadboys, Howard L. (with Harrison, James L., and Litwak, Robert S.), Direct vision approach to aortic valve without occlusion or bypass, 756

Gebauer, Paul W. (with Thomas, Paul A.), Pleurectomy for recurrent spontaneous pneumothorax, 111

Geisler, James W. (with Webb, Watts R., and Johnston, J. Harvey, Jr.), Pneumomediastinum: physiologic observations, 309

Gilman, Richard A. (with Bailey, Charles P., Bolton, Houck E., and Nichols, Henry), Ventriculoplasty for cardiac aneurysm, 37

Gladding, Thomas C. (with Edwards, Jesse E., and Weir, Alva B., Jr.), Congenital communication between right coronary artery and right atrium, 662

Glover, Robert P. (with Davila, Julio C., Voci, Gerardo, Jumbala, P., Trout, Robert G., and Fritz, A. Jane), Clinical and physiologic criteria for surgical correction of mitral insufficiency, 206

Golberg, Mauricio (with Patt, Howard H., Clift, John B., and Lourvanij, Bunket), Evaluation of implantation of internal mammary artery into myocardium of pigs and dogs, 699

Gold colloid, radioactive silver-coated, administered intrathoracically to dogs, further observations on the lymphatic pick-up of (Matuska, Hahn, Carlson, Auerbach, and Meneely), 135

Goldman, Alfred, Discussion of ventriculoplasty for cardiac aneurysm, 66

Gott, Vincent L. (with Lillehei, C. Walton, DeWall, Richard A., and Varco, Richard L.), Surgical treatment of stenotic or regurgitant lesions of mitral and aortic valves by direct vision utilizing pump-oxygenator, 154

Grajo, Maria Z. (with Lurie, Paul R.), Resistance to catheter puncture of various cardiovascular structures, 751

Granular-cell myoblastoma (organoid type) involving arm, lung, and brain, with twenty years' survival, case of (*Meredith, Kay, and Bosher*), 80

Granulomata of mediastinum surgically treated and followed up to nine years (Karlson and Timmes), 617

Grillo, Hermes C. (*with Scannell, J. Gordon*), Primary tumors of heart, 23

H

Hahn, P. F. (with Matuska, R. A., Carlson, R. I., Auerbach, S. H., and Meneely, G. R.), Further observations on lymphatic pick-up of radioactive silver-coated gold colloid administered intrathoracically to dogs, 135

Hamartoma of lung (Cavin, Masters, and Moody), 816

Hanlon, Rollins, Discussion of primary tumors of heart, 34

Hannon, Donald M. (with Lewis, F. John), Compact suction apparatus for chest drainage, 696

Harken, Dwight E. (with Taylor, Warren J., Thrower, Wendell B., and Black, Harrison), Surgical correction of aortic insufficiency by circumclusion, 192

Harrison, James L. (with Gadboys, Howard L., and Litwak, Robert S.), Direct vision approach to aortic valve without occlusion or bypass, 756

Harte, Maurice (with Aufses, Arthur H.), Extrapleural thoracoplasty in treatment of pulmonary tuberculosis: an eight- to twenty-one-year follow-up study, 332

Hartley, Lawrence J. (with Sweetman, William R., Bauer, Albert J., and Salyer, John M.), Postinflammatory "tumor" of lung, 802

Hausmann, Paul F., Pulmonary aspergilloma, 538

Heart, primary tumors of (Scannell and Grillo), 23

Hearts with interventricular septal defects, conduction system in (Truex and Bishof), 421

Helmholz, H. Frederic, Jr. (with Miller, R. Drew, Bridge, Ezra V., Jr., Fowler, Ward S., Ellis, F. Henry, Jr., and Allen, George T.), Pulmonary function before and after pulmonary resection in tuberculous patients, 651

Hennigar, Gordon R. (with Choy, S. H.), Accessory lung with persistent left superior vena cava and duplication of intestine, 469

Massarelli, L G (with Antonius, N A, and Crecca, A D), Clinical evaluation of surgical treatment of thirty two cases of coronary artery disease, 68

Masters, Joseph H (with Cavin, Elwyn, and Moody, James), Hamartoma of lung, 816

Matas, Rudolph, In memoriam, 1

Matuska, R A (with Hahn, P F, Carlson R I, Auerbach, S H, and Meneely, G R), Further observations on lymphatic pick up of radioactive silver coated gold colloid administered intrathoracically to dogs, 135

McGoon, Dwight C (with Kirklin, John W), Surgical technique for repair of high ventricular septal defects, 584

McIntyre, David (with Smith, Franklin R), Ivalon graft to bridge coronary artery occlusion, 513

McKnight, Evelyn (with Ayes, Fred H, and Leidler, Franz), Right left identification of bronchoscopy smears, 829

Mediastinal granulomas revised concept of their incidence and etiology (Peabody, Brown, Sullivan, and Cannon), 384

Mediastinum, granulomata of, surgically treated and followed up to nine years (Karlson and Timmes), 617

  pancreatic pseudocyst of (Clauss and Wilson), 795

  primary extragenital chorioepithelioma in the male (Magovern and Blades), 378

  secondary neoplasms of (Sanborn, Beattie, and Slaughter), 678

  tuberculoma of report of first culturally proved case (Peabody, Walkup, and Murphy), 397

Meneely, G R (with Matuska, R A, Hahn, P F, Carlson, R I, and Auerbach, S H), Further observations on lymphatic pick up of radioactive silver coated gold colloid administered intrathoracically to dogs, 135

Meredith, J M (with Kay, Saul, and Bosher, Lewis H, Jr), Case of granular cell myoblastoma (organoid type) involving arm, lung, and brain, with twenty years' survival, 80

Metastasis of two tumors, three primary carcinomas of lung arising in left lower lobe with, case report (Newman and Adkins), 474

Meyer, Kenneth K, Direct lymphatic connections from lower lobes of lung to abdomen, 726

Miller, R Drew (with Bridge, Ezra V, Jr, Fowler, Ward S, Helmholz, H Frederic, Jr, Ellis, F Henry, Jr, and Allen, George T), Pulmonary function before and after pulmonary resection in tuberculous patients, 601

Mitral and aortic valves by direct vision utilizing pump oxygenator, surgical treatment of stenotic or regurgitant lesions of (Lillehei, Gott, DeWall, and Varco), 154

  insufficiency, surgical correction of, by use of pericardial valve graft (Bakst, Boley, Joseph, and Loewe), 492

    clinical and physiologic criteria for surgical correction of (Davila, Glover, Voci, Jumbala, Trout, and Fritz), 206

Moody, James (with Cavin, Elwyn, and Masters, Joseph H), Hamartoma of lung, 816

Morphine and amiphenazole for postoperative analgesia following major thoracic surgery (Weber), 105

Morse, Dryden P (with Hirose, Teruo, and Bailey, Charles P), Atrioseptopexy simplified new technique, 255

Mucormycosis of bronchus successfully treated by lobectomy (Dillon, Sealy, and Fetter), 464

Mulhim, Rafik (with Siouji, Maurice, and Wilson, John L), Hydatid cyst of lung with bronchographic evaluation of treatment by internal suture of pericyst, 779

Murphy, James D (with Peabody, J Winthrop, Jr, and Walkup, Harry E), Tuberculoma of mediastinum report of first culturally proved case, 397

Musser, Benjamin G (with Bailey, Charles P), Fenestrated cardiovascular clamps, 549

Myers, Myron J (with Nachlas, Marvin M, Solomon, Robert D, Kelly, Alma B, and Seligman, Arnold M), Maintenance of patency of carotid arteries implanted into myocardium, 706

Myoblastoma, granular cell (organoid type) involving arm, lung and brain, with twenty years' survival (Meredith, Kay, and Bosher), 80

Myocardium, maintenance of patency of carotid arteries implanted into (Nachlas, Myers, Solomon, Kelly, and Seligman), 706

  of pigs and dogs, evaluation of implantation of internal mammary artery int (Patt, Golberg, Clift, and Lourvanij), 699

N

Nachlas, Marvin M (with Myers, Myron J, Solomon, Robert D, Kelly, Alma B, and Seligman, Arnold M), Maintenance of patency of carotid arteries implanted into myocardium, 706

Ness, Delbert D (with Knuth, Warren P, and Walske, Benedict R), Coarctation of aorta with report of unusual o two [illegible] cation, 591

Kurzweg, Frank T. (with Fomon, John J., Davis, Harold, Broadaway, Rufus K., and Chesney, John), Single dosage x-ray radiation to lung and *posterior mediastinum through open* chest. I. Results in dogs receiving dosage between 6,000 and 15,000 r, 771

L

Latson, Joseph R. (with Cooley, Denton A., Belmonte, Benjamin A., and Pierce, James F.), Bubble diffusion oxygenator for cardiopulmonary bypass, 131

Leidler, Franz (with Aves, Fred H., and McKnight, Evelyn), Right-left identification of bronchoscopy smears, 829

Leiomyosarcoma, pneumonectomy for (Ochsner and Ochsner), 768

Lemmon, W. M. (with Bailey, Charles P.), New classification for coarctations of aorta, 291

Lewis, F. John (with Hannon, Donald M.), Compact suction apparatus for chest drainage, 696

Lillehei, C. Walton (with Gott, Vincent L., DeWall, Richard A., and Varco, Richard L.), Surgical treatment of stenotic or regurgitant lesions of mitral and aortic valves by direct vision utilizing pump-oxygenator, 154, 191

Linder, Erland (with Bergman, Frank), Tumor-forming amyloidosis of lung, 628

Lipomas of bronchus (Hutcheson, Ashe, and Paulson), 638

Litwak, Robert S. (with Gadboys, Howard L., and Harrison, James L.), Direct vision approach to aortic valve without occlusion or bypass, 756

Lobectomy, carcinoma of lung treated by; problem of local recurrence (Holland), 274
  mucormycosis of bronchus successfully treated by (Dillon, Sealy, and Fetter), 464

Loewe, Leo (with Bakst, Alvin A., Boley, Scott J., and Joseph, Edward), Surgical correction of mitral insufficiency by use of pericardial valve graft, 492

Lourvanij, Bunket (with Patt, Howard H., Golberg, Mauricio, and Clift, John V.), Evaluation of implantation of internal mammary artery into myocardium of pigs and dogs, 699

Lueders, H. W. (with Stranahan, Allan, Alley, R. D., Kausel, H. W., and Peck, A. S.), Simplified method for achieving intrarespiratory humidification in tracheotomized patient, 461

Lundberg, Åke, Intracardiac pressure measurement during operation for congenital heart disease, 740

Lung accessory, with persistent left superior vena cava and duplication of intestine (Hennigar and Choy), 469
  direct lymphatic connections from lower lobe to abdomen (Meyer), 726
  hamartoma of (Cavin, Masters, and Moody), 816
  and posterior mediastinum through the open chest, single dosage x-ray radiation to. I. Results in dogs receiving dosage between 6,000 and 15,000 r (Fomon, Davis, Kurzweg, Broadaway and Chesney), 771
  postinflammatory "tumor" of (Sweetman, Hartley, Bauer, and Salyer), 802
  tumor-forming amyloidosis of (Bergman and Linder), 628

Lurie, Paul R. (with Grajo, Maria Z.), Resistance to catheter puncture of various cardiovascular structures, 751

Lymphatic connections, direct, from lower lobes of lung to abdomen (Meyer), 726
  pick-up of radioactive silver-coated gold colloid administered intrathoracically to dogs, further observations on (Matuska, Hahn, Carlson, Auerbach, and Meneely), 135

M

Magistro, Roberto (with Robicsek, Francis, Foti, Egidio, Robicsek, Livia, and Sanger, Paul W.), Vena cava—pulmonary artery anastomosis for vascularization of lung, 440

Magovern, George J. (with Blades, Brian), Primary extragenital chorioepithelioma in male mediastinum, 378

Mahoney, Earle B., Discussion of reconstruction of esophagus with segments of colon, 19

Maisel, Bernard (with Steinberg, Israel, and Vogel, F. Stephen), Pulmonary arteriovenous fistula associated with capillary telangiectasis (Rendu-Osler-Weber disease): report of case illustrating use of metal casting for demonstrating lesion, 517

Mangiardi, J. L., Discussion of ventriculoplasty for cardiac aneurysm, 64

Mannheimer, E. (with Brynolf, I., and Crafoord, C.), Coarctation of aorta proximal to both subclavian arteries, 123

Mannix, Edgar P., Jr., Discussion of primary tumors of heart, 34

Marcus, Aaron J. (with Spaet, Theodore H.), Ineffectiveness of Adrenosem in pulmonary surgery, 821

Promethazine hydrochloride in bronchoscopy, amnesic effect of (Pino and Van Houten), 825

Pulmonary arteriovenous fistula associated with capillary telangiectasia (Rendu Osler Weber disease) report of case illustrating use of metal casting for demonstrating lesion (Steinberg, Maisel, and Vogel), 517

- artery—vena cava anastomosis for vascularization of lung (Robicsek, Magistro, Foti, Robicsek, and Sanger), 440
  - occlusion in man, unilateral II Studies in patients with chronic pulmonary disease (Charms, Brofman, Elder, and Kohn), 316
- aspergilloma (Hausmann), 538
- aspergillosis, case report (Foushee and Norris), 542
- carcinomas, nonresectable, angiocardiography as aid to identification of (Wyman and Wilkins), 452
- cryptococcosis (torulosis), resection in (White and Arany), 402
- function before and after pulmonary resection in tuberculous patients (Miller, Bridge, Fowler, Helmholz, Ellis, and Allen), 651
- hemorrhage in tuberculosis, the surgical treatment of (Ford, Clauss, Askari, and Wylie), 341
- surgery, ineffectiveness of Adrenosem in (Marcus and Spaet), 821
- tuberculosis, extrapleural thoracoplasty in treatment of an eight to twenty-one year follow up study (Harte and Aufses), 332
- valvular insufficiency, experimental, effect on circulation (Fowler and Duchesne), 643

Pump oxygenator, surgical treatment of stenotic or regurgitant lesions of mitral and aortic valves by direct vision utilizing a (Lillehei, Gott, DeWall, and Varco), 154

- use of a modified Gibbon type (Diettert, Bercu, and Ferguson), 416

R

Radiation therapy for recurrent carcinoma in bronchial stump (Pinsky and Emerson), 683

- to lung and posterior mediastinum through open chest, single dosage x ray I Results in dogs receiving dosage between 6 000 and 15,000 r (Fomon, Davis, Kurzweg, Broadaway, and Chesney), 771

Rainer, W G (with Eiseman, B), Clinical management of posttraumatic rupture of thoracic aorta, 347

Robertson, Ross, Discussion of primary tumors of heart, 35

Robicsek, Francis (with Magistro, Roberto, Foti, Egidio, Robicsek, Livia, and Sanger, Paul W), Vena cava—pulmonary artery anastomosis for vascularization of lung, 440

Robicsek, Livia (with Robicsek, Francis Magistro, Roberto, Foti, Egidio, and Sanger, Paul W), Vena cava—pulmonary artery anastomosis for vascularization of lung, 440

Rodriguez, Louis E (with Bjork , Viking Olov), Reconstruction of trachea and its bifurcation, 596

Roe, Benson B , Discussion of papers on correction of aortic insufficiency and surgical correction of mitral insufficiency, 230

S

Sabiston, David C , Jr , Surgical management of congenital bifid sternum with partial ectopia cordis, 118

Šalek J (with Pazderka, S and Žak , F), Solitary bronchial polyps of inflammatory origin, 807

Salyer, John M (with Sweetman, William R , Hartley, Lawrence J , and Bauer, Albert J) Postinflammatory "tumor" of lung, 802

Sanborn, E B (with Beattie, E J Jr , and Slaughter, D P), Secondary neoplasms of mediastinum, 678

Sanger, Paul W (with Robicsek, Francis, Magistro, Roberto Foti, Egidio, and Robicsek, Livia), Vena cava—pulmonary artery anastomosis for vascularization of lung, 440

Scannell, J Gordon (with Grillo, Hermes C), Primary tumors of heart, 23, 36

Scapular traction for closure of lateral or posterolateral chest incisions, simple method of (Brantigan and de Borja), 400

Schobinger, Robert, Internal mammary venography, 692

Sealy, W C (with Dillon, M L , and Fetter B F), Mucormycosis of bronchus successfully treated by lobectomy, 464

Seligman, Arnold M (with Nachlas Marvin M , Myers, Myron J , Solomon, Robert D , and Kelly, Alma B), Maintenance of patency of carotid arteries implanted into myocardium, 706

Septal defects, surgical technique for repair of high ventricular (Kirklin and McGoon), 584

- leaflet, rehinging of, maximal reconstitution of stenotic mitral valve by (neostrophingic mobilization) (Bailey and Hirose), 579

Neoplasms of mediastinum, secondary (Sanborn, Beattie, and Slaughter), 678

Neostrophingic mobilization (rehinging of septal leaflet), maximal reconstitution of stenotic mitral valve by (Bailey and Hirose), 559

Neville, William E. (with Clowes, George H. A., Jr.), Reconstruction of esophagus with segments of colon, 2, 22

Newman, William (with Adkins, Paul C.), Three primary carcinomas of lung arising in left lower lobe with metastasis of two of the tumors, 474

Nichols, Henry (with Bailey, Charles P., Bolton, Houck E., and Gilman, Richard A.), Ventriculoplasty for cardiac aneurysm, 37

Norris, Franklin G. (with Foushee, J. H. Smith), Pulmonary aspergillosis; case report, 542

O

Ochsner, Alton (with Ochsner, Seymour), Pneumonectomy for leiomyosarcoma, 768

Ochsner, Seymour (with Ochsner, Alton), Pneumonectomy for leiomyosarcoma, 768

Okano, Takeshi (with Walkup, Harry E.), Some problems in extrapleural pneumonectomy for tuberculous empyema and destroyed lung, 523

Overton, Robert C. (with Creech, Oscar), Unusual esophageal atresia with distant membranous obstruction of esophagus, 674

Oxygenator, bubble diffusion, for cardiopulmonary bypass (Cooley, Belmonte, Latson, and Pierce), 131
  stainless steel bubble (Beretta, Jordan, and Tolstedt), 411
  use of modified Gibbon-type pump (Diettert, Bercu, and Ferguson), 416

P

Pancreatic pseudocyst of mediastinum (Clauss and Wilson), 795

Patt, Howard H. (with Golberg, Mauricio, Clift, John B., and Lourvanij, Bunket), Evaluation of implantation of internal mammary artery into myocardium of pigs and dogs, 699

Paulson, Donald L. (with Hutcheson, James B., and Ashe, William M.), Lipomas of bronchus, 638

Pazderka, S. (with Šálek, J., and Žák, F.), Solitary bronchial polyps of inflammatory origin, 807

Peabody, J. Winthrop, Jr. (with Brown, Robert B., Sullivan, M. Bruce, and Cannon, Albert), Mediastinal granulomas: revised concept of their incidence and etiology, 384

Peabody, J. Winthrop, Jr.—Cont'd
  (with Walkup, Harry E., and Murphy, James D.), Tuberculoma of mediastinum; report of first culturally proved case, 397

Peck, A. S. (with Lueders, H. W., Stranahan, Allan, Alley, R. D., and Kausel, H. W.), Simplified method for achieving intrarespiratory humidification in tracheotomized patient, 461

Pectus excavatum, surgical treatment of (Daniel), 719

Pence, Robert E. (with Hyun, Bong Hak, and Dawson, Elizabeth A.), Bacterial and mycotic endocarditis following cardiac surgery, 298

Pericardial fat necrosis: an unusual entity (Kasserman), 689
  valve graft, surgical correction of mitral insufficiency by use of (Bakst, Boley, Joseph, and Loewe), 492

Perkins, Rex B. (with Dillard, Richard A.), Spontaneous bilateral chylopneumothorax, 91

Pierce, James F. (with Cooley, Denton A., Belmonte, Benjamin A., and Latson, Joseph R.), Bubble diffusion oxygenator for cardiopulmonary bypass, 131

Pino, D. M. (with Van Houten, R. J.), Amnesic effect of promethazine hydrochloride in bronchoscopy, 825

Pinsky, Harry J. (with Emerson, George L.), Radiation therapy for recurrent carcinoma in bronchial stump, 683

Pleurectomy for recurrent spontaneous pneumothorax (Thomas and Gebauer), 111

Pneumomediastinum: physiologic observations (Webb, Johnston, and Geisler), 309

Pneumonectomy, extrapleural, for tuberculous empyema and destroyed lung, some problems in (Okano and Walkup), 523
  for leiomyosarcoma (Ochsner and Ochsner), 768

Pneumothorax, spontaneous, pleurectomy for recurrent (Thomas and Gebauer), 111

Postinflammatory "tumor" of lung (Sweetman, Hartley, Bauer, and Salyer), 802

Posttraumatic rupture of thoracic aorta, clinical management of (Eiseman and Rainer), 347

Pressure-flow relationships of Sigmamotor pump as used in cardiac bypass (Shimomura, Andreae, Bell, and Fitzpatrick), 747

Primary extragenital chorioepithelioma in male mediastinum (Magovern and Blades), 378

Trout, Robert G (with Davila, Julio C, Glover Robert P, Voci, Gerardo, Jumbala, P, and Fritz A Jane), Clinical and physiologic criteria for surgical correction of mitral insufficiency, 206

Truex, Raymond C (with Bishof, Josephine Kain), Conduction system in human hearts with interventricular septal defects, 421

Tuberculoma of mediastinum report of first culturally proved case (Peabody Walkup, and Murphy 397

Tuberculosis, *surgical treatment of pulmonary* hemorrhage in (Lord Claus Aikawa and Wylie), 141

Tuberculous empyema and destroyed lung, some problems in extrapleural pneu*monectomy for (Okano and Walk*up), 523
  patients, pulmonary function before and after pulmonary resection in (Miller *Bridge Fowler Helmholz, Ellis and* Allen), 651

Tulanowicz, S Anthony (with Paret Alexander C, DeLong Robert P and Blakemore William S) Transfixion of aorta accompanied by Brown Sequard syndrome 3[illegible]9

Tumor forming amyloidosis of lung *(Bergman and Linder), 628*

Tumors of esophagus, benign, pedunculated intraluminal (Bernatz, Smith Ellis, and Andersen) 303
  primary, of heart (Scannell and Grillo), 23

V

Van Houten, R T (with Pino, D M), *Amnesic effect of promethazine hy*drochloride in bronchoscopy, 825

Varco, Richard L (with Lillehei, C Walton Gott, Vincent L , and DeWall, Richard A), Surgical treatment of stenotic or regurgitant lesions of mitral and aortic valves by direct vision utilizing pump oxygenator, 154

Vascularization of lung, vena cava—pulmo*nary artery anastomosis* for (Robicsek, Magistro, Foti, Robicsek, and *Sanger), 440*

Vena cava, persistent left superior, and accessory lung with duplication of intestine (Hennigar and Choy) 469
  —pulmonary artery anastomosis for vascularization of lung (Robicsek, Magistro, Foti, Robicsek, and Sanger), 440

Venography, internal mammary (Schobinger), *692*

Ventricular diastolic pressure, effect of inflow obstruction and rapid *bleeding* on (Fowler, Couves, and Bewick) 532

Ventricular—*Cont'd*
  fibrillation following hypothermia and induced cardiac arrest, experimental prevention of (Berman, Taylor, and Fisch) 483
    in hypothermia (Badeer) 265
  septal defects, conduction system in human hearts with (Truex and Bishof), 421

Ventriculoplasty for cardiac aneurysm *(Bailey, Bolton,* Nichols, and Gilman), 37

Vineberg Arthur Discussion of coronary artery disease 254

Voci, Gerardo (with Davila Julio C, Glover, Robert P Jumbala P, Trout, Robert G, and Fritz, A Jane), Clinical and physiologic criteria for surgical correction of mitral insufficiency, 206

Vogel, F Stephen *(with Steinberg, Israel,* and Mussel, Bernard), Pulmonary *arteriovenous fistula associated with* capillary telangiectasia (Rendu Osler Weber disease) report of case illustrating use of metal casting for demonstration lesion 517

W

Walkup Harry E (with Okano, Takeshi), *Some problems in extrapleural pneu*monectomy for tuberculous empyema and destroyed lung, 523
  (with Peabody, J Winthrop, Jr , and Murphy James D) Tuberculoma of mediastinum, report of first culturally proved case, 397

Walske, *Benedict R* (with Knuth, Warren P , and Neis, Delbert D) Coarctation of aorta with report of unusual operative complication, 591

Wangensteen Owen H , Discussion of reconstruction of esophagus with segments of colon, 20

Webb Watts R (with *Johnston, J Harvey,* Jr , and Geisler, James W), Pneumomediastinum physiologic *observations*, 309

Weber, *T C P*, Morphine and amiphenazole for postoperative analgesia following major thoracic surgery, 105

Weir, Alva B *Jr* (with Edwards, Jesse E , and Gladding, Thomas C), Congenital *communication* between right *coronary artery and right atrium, 662*

White, Manuel (with Arany, L S), Resection in pulmonary cryptococcosis (torulosis), 402

Wilkins, Earle W , Jr (with Wyman, Stanley M), Angiocardiography as aid to identification of nonresectable pulmonary carcinomas, 452

Sherman, Paul H. (with Conant, James S.), Bronchial closure with catgut sutures, 363

Shimomura, Seiichi (with Andreae, Eric, Bell, A. L. Loomis, Jr., and Fitzpatrick, Hugh F.), Pressure-flow relationships of Sigmamotor pump as used in cardiac bypass, 747

Shumacker, Harris B. (with King, Harold), Surgical repair of traumatic aortic-right ventricular fistula, 734

Sigmamotor pump as used in cardiac bypass, pressure-flow relationships of (Shimomura, Andreae, Bell, and Fitzpatrick), 747

Slaughter, D. P. (with Sanborn, E. B., and Beattie, E. J., Jr.), Secondary neoplasms of mediastinum, 678

Sleeve resection of main-stem bronchus, bronchial adenoma treated by (Connolly and Chamberlain), 372

Smith, Franklin R. (with McIntyre, David), Ivalon graft to bridge coronary artery occlusion, 513

Smith, J. Lawrence (with Bernatz, Philip E., Ellis, F. Henry, Jr., and Andersen, Howard A.), Benign, pedunculated, intraluminal tumors of esophagus, 503

Solomon, Robert D. (with Nachlas, Marvin M., Myers, Myron J., Kelly, Alma B., and Seligman, Arnold M.), Maintenance of patency of carotid arteries implanted into myocardium, 706

Spaet, Theodore H. (with Marcus, Aaron J.), Ineffectiveness of Adrenosem in pulmonary surgery, 821

Srouji, Maurice (with Mulhim, Rafik, and Wilson, John L.), Hydatid cyst of lung with bronchographic evaluation of treatment by internal suture of pericyst, 779

Steinberg, Israel (with Maisel, Bernard, and Vogel, F. Stephen), Pulmonary arteriovenous fistula associated with capillary telangiectasia (Rendu-Osler-Weber disease): report of case illustrating use of metal casting for demonstrating lesion, 517

Stenotic mitral valve, maximal reconstitution by neostrophingic mobilization (rehinging of septal leaflet), (Bailey and Hirose), 559

Stranahan, Allan (with Lueders, H. W., Alley, R. D., Kausel, H. W., and Peck, A. S.), Simplified method for achieving intrarespiratory humidification in tracheotomized patient, 461

Suction apparatus, compact, for chest drainage (Lewis and Hannon), 696

Sullivan, M. Bruce (with Peabody, J. Winthrop, Jr., Brown, Robert B., and Cannon, Albert), Mediastinal granulomas, revised concept of their incidence and etiology, 384

Swan, Henry (with Wilkinson, Robert H., and Blount, S. Gilbert, Jr.), Visual repair of congenital aortic stenosis during hypothermia, 139

Sweet, Richard H., Discussion of reconstruction of esophagus with segments of colon, 22

Sweetman, William R. (with Hartley, Lawrence J., Bauer, Albert J., and Salyer, John M.), Postinflammatory "tumor" of lung, 802

T

Taylor, Max T. (with Berman, Edward J., and Fisch, Charles), Experimental prevention of ventricular fibrillation following hypothermia and induced cardiac arrest, 483

Taylor, Warren J., Discussion of papers on correction of aortic insufficiency and surgical correction of mitral insufficiency, 231
  (with Thrower, Wendell B., Black, Harrison, and Harken, Dwight E.), Surgical correction of aortic insufficiency by circumclusion, 192

Thomas, George I., Discussion of reconstruction of esophagus with segments of colon, 19

Thomas, Paul A. (with Gebauer, Paul W.), Pleurectomy for recurrent spontaneous pneumothorax, 111

Thoracic aorta, clinical management of post-traumatic rupture of (Eiseman and Rainer), 347

Thoracoplasty, extrapleural, in treatment of pulmonary tuberculosis: eight- to twenty-one-year follow-up study (Harte and Aufses), 332

Thoracotomy, experimental bilateral transsternal, factors improving survival (Kortz), 305

Thrower, Wendell B. (with Taylor, Warren J., Black, Harrison, and Harken, Dwight E.), Surgical correction of aortic insufficiency by circumclusion, 192

Timmes, Joseph J. (with Karlson, Karl E.), Granulomata of mediastinum surgically treated and followed up to nine years, 617

Tolstedt, Grandon E. (with Jordan, Prescott, Jr., and Beretta, Francesco F.), Stainless steel bubble oxygenator, 411

Trachea and its bifurcation, reconstruction of (Björk and Rodriguez), 596

Tracheobronchial rests in esophagus (Bergmann and Charnas), 97

Tracheotomized patient, simplified method for achieving intrarespiratory humidification in (Lueders, Stranahan, Alley, Kausel, and Peck), 461

Traumatic aortic-right ventricular fistula, surgical repair of (King and Shumacker), 734

Wilkinson, Robert H. (with Swan, Henry, and Blount, S. Gilbert, Jr.), Visual repair of congenital aortic stenosis during hypothermia, 139

Wilson, David W. (with Clauss, Roy H.), Pancreatic pseudocyst of mediastinum, 795

Wilson, John L. (with Srouji, Maurice, and Mulhim, Rafik), Hydatid cyst of lung with bronchographic evaluation of treatment by internal suture of pericyst, 779

Wound surgery encounters a dilemma (Churchill), 279

Wylie, Robert H. (with Ford, Joseph M., Clauss, Roy H., and Askari, Salah Al), Surgical treatment of pulmonary hemorrhage in tuberculosis, 341

Wyman, Stanley M. (with Wilkins, Earle W., Jr.), Angiocardiography as aid to identification of nonresectable pulmonary carcinomas, 452

Z

Žák, F. (with Šálek, J., and Pazderka, S.) Solitary bronchial polyps of inflammatory origin, 807